*A DISSERTATION ON*

# A CLINICAL STUDY ON GASTRIC OUTLET OBSTRUCTION IN 50 CASES

*Dissertation Submitted to*

**THE TAMILNADU**
**DR.M.G.R. MEDICAL UNIVERSITY**
**CHENNAI – 600 032**

***With fulfillment of the Regulations***
***For the Award of the Degree of***

**M.S. GENERAL SURGERY**
**Branch - I**


**DEPARTMENT OF GENERAL SURGERY**
**KILPAUK MEDICAL COLLEGE**

**CHENNAI – 600 010**

**MARCH - 2009**

# CERTIFICATE

This in to certify that the dissertation work titled **"A CLINCAL STUDY ON GASTRIC OUTLET OBSTRUCTION IN 50 CASES"** is a bonafide research work of Dr. V. S. **Valarmathy**, Enrolment No.

submitted in partial fulfillment of the requirements for the award of Degree of **M.S. GENERAL SURGERY** in THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY CHENNAI- 600 032

Signature of unit Chief Signature of H.O.D

Signature of Dean

# ACKNOWLEDGEMENT

I am greatly indebted to my **Chief Prof. T. BAVANISANKAR, M.S.,** for his valuable guidance and contribution in completing this study.

I am grateful to the **DEAN, DME (OSD) Prof. Dr. M. DHANAPAL,** Kilpauk medical college and **Prof. G. GUNASEELAN M.S** Head of the Department of surgery for permitting me to utilize the clinical material in KMCH Chennai, during the period of my study.

I am thankful to my Assistant professors Dr. **V.RUKMANGATHAN, M.S. and Dr. M. ALLI, M.S, Dr. P.S. SHANTHI M.S** clarifying all my doubts and for putting forth all their efforts to make this study a complete one.

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# CONTENTS

# *INTRODUCTION*

# INTRODUCTION

Gastric outlet obstruction one of the most common problem encountered in General Surgery. The causes behind this common problem has changed, comparing with the incidence in the past.

In this study on the gastric outlet obstruction, the various aspects and management of the gastric outlet obstruction, are analysed. In this study, main focus is on the two most common causes of gastric outlet obstruction and their management in the Government kilpauk medical college hospital

# AIM OF STUDY

# AIM OF THE STUDY

1. To study the etiological causes of gastric outlet obstruction in adults getting admitted in Government Kilpauk Medical College Hospital

2. To study sex and age distribution in each of the causes.

3. To study the various types of management

4. To find the most commonly performed surgeries for the two most common causes.

# MATERIALS

# AND

# METHODS USED

# MATERIAL AND METHODS USED

This study on Gastric outlet obstruction is based on a study of 50 cases that were admitted in Government kilpauk Hospital, Chennai during the period of October 2006 to October 2008

## EXCLUSION CRITERIA FOR THE PATIENTS

1. Patients younger than 18 years of age

2. Patients with preadmission diagnosis of carcinoma stomach / chronic duodenal ulcer through endoscopy done outside.

The patients were investigated by routine investigations like upper gastrointestinal endoscopy, ultrasonography of the abdomen, barium meal and CT abdomen in selected cases.

All cases were taken up for curative/ palliative surgery and operated depending upon the causes

# REVIEW

# OF

# LITERATURE

# REVIEW OF LITERATURE

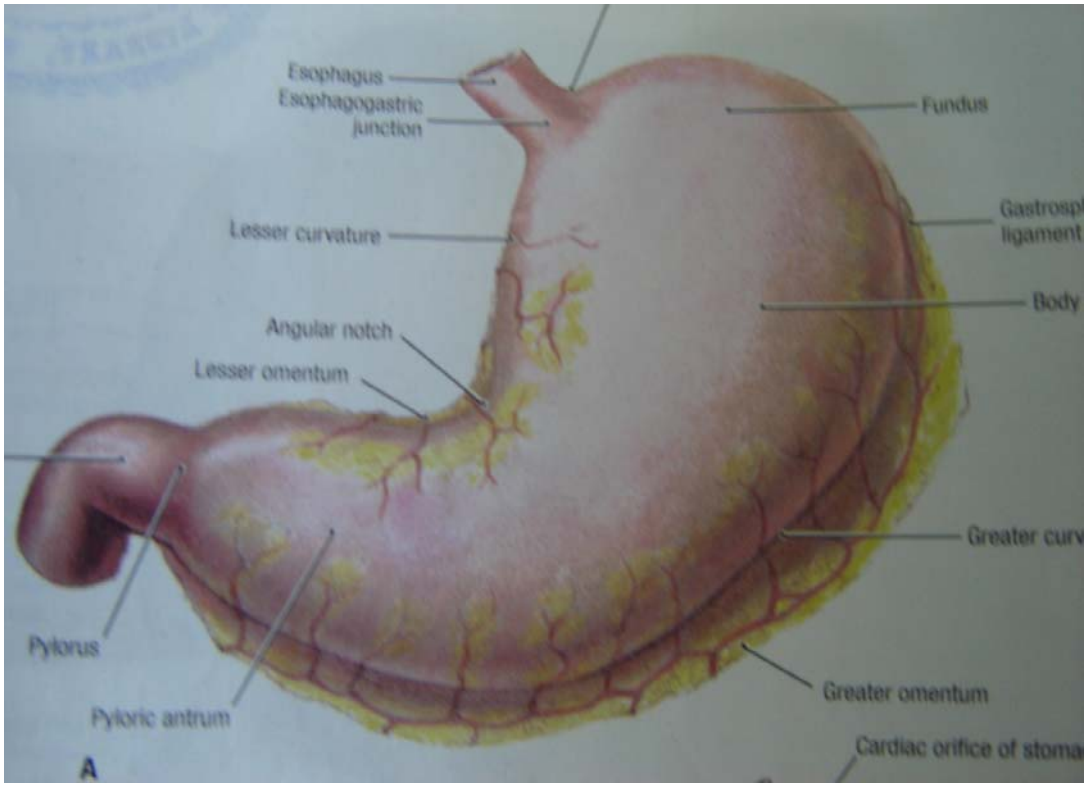
## ANATOMY

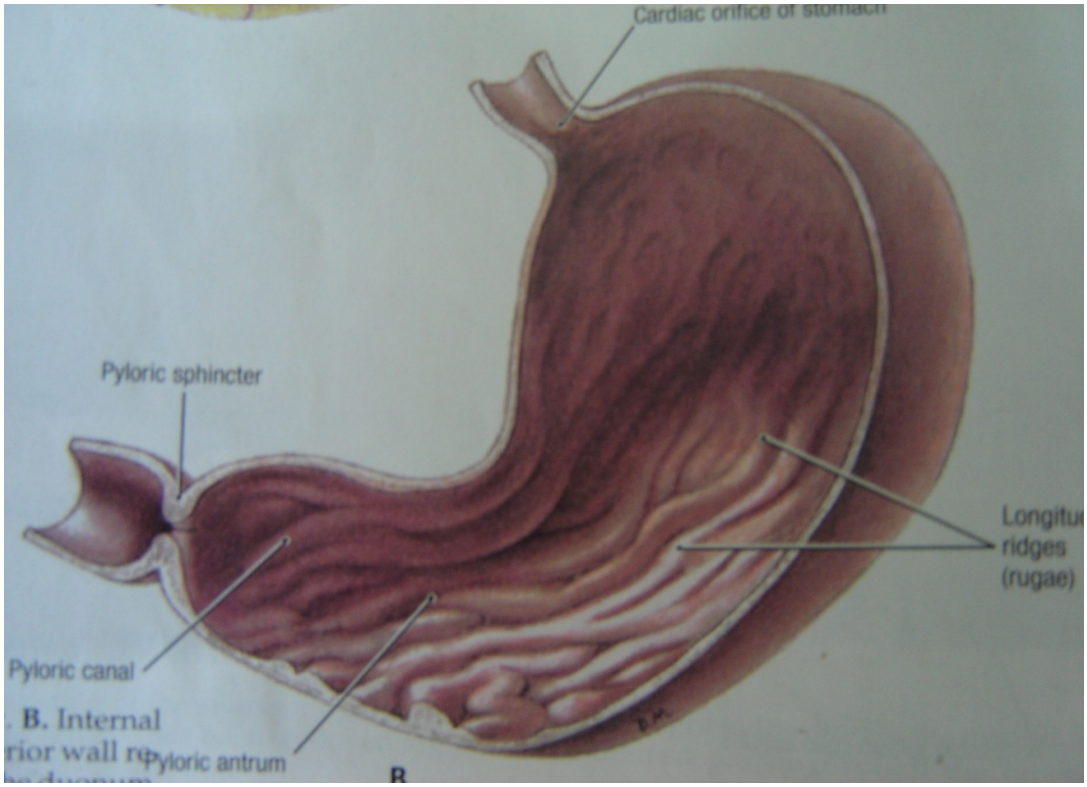
The stomach is the most dilated part of the alimentary tract, interposed between the esophagus and the duodenum in the upper part of the abdominal cavity and lying mainly in the left hypochondrium, epigastric and umbilical region with much of it under the cover of lower ribs. It is a muscular bag relatively fixed at both the ends. The stomach is curved anteriorly. The stomach is subjected to great variation in size depending on the volume of it's content.

## ORIFICES OF STOMACH

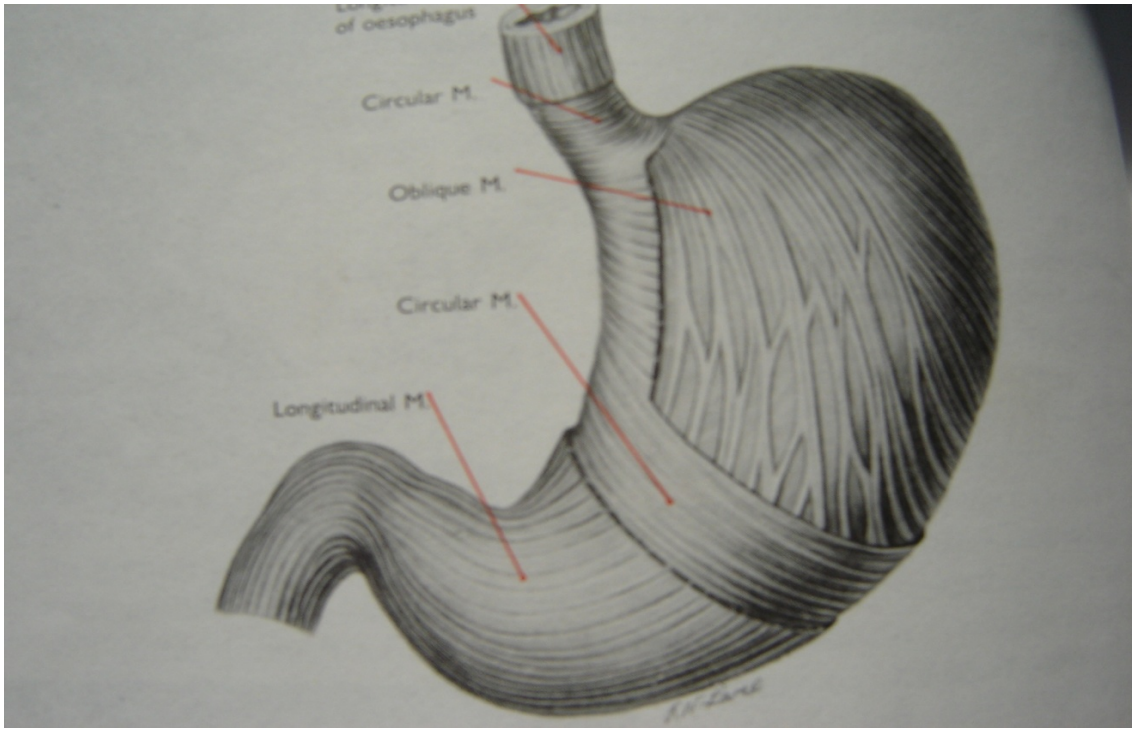
The cardiac orifice is at the Gastro esophageal junction, 40cm from the incisor teeth. It is the most fixed part of the organ. It is 2.5 cm to left of the midline at level of $T_{10}$ vertebrae. At the distal end is the pylorus. This is relatively mobile than cardia.

# ANATOMY OF STOMACH

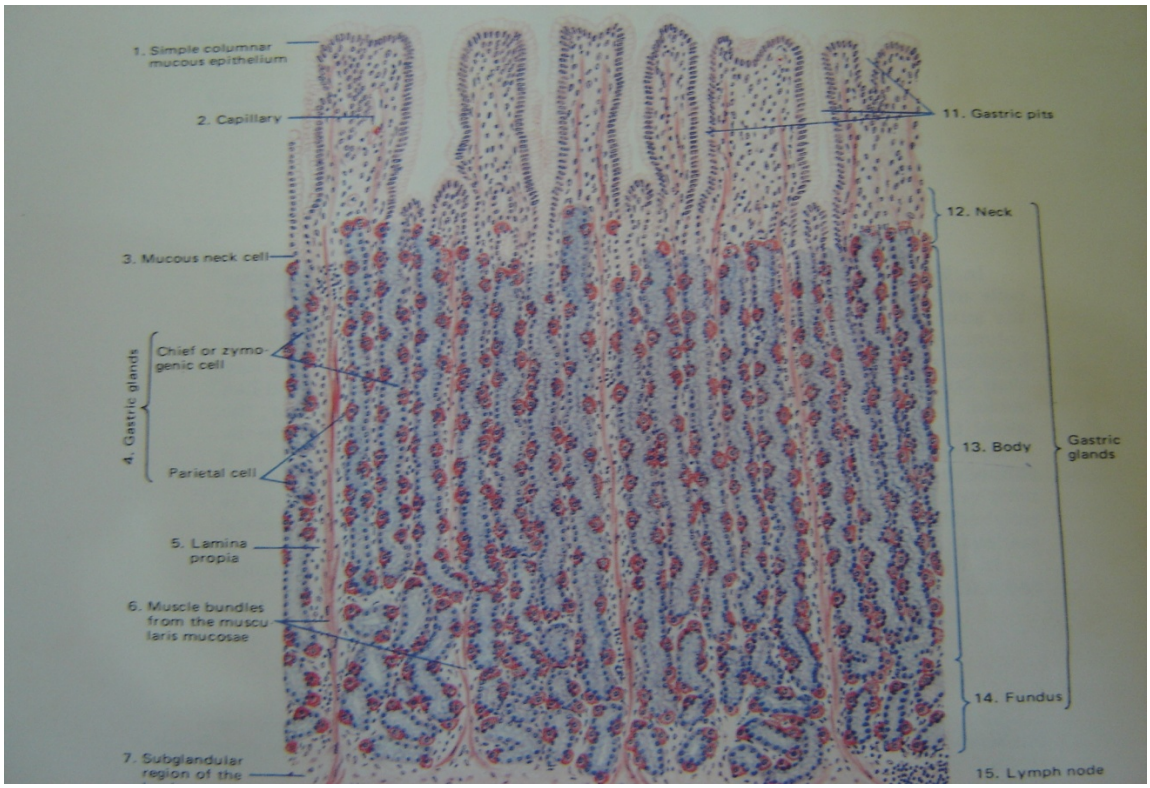


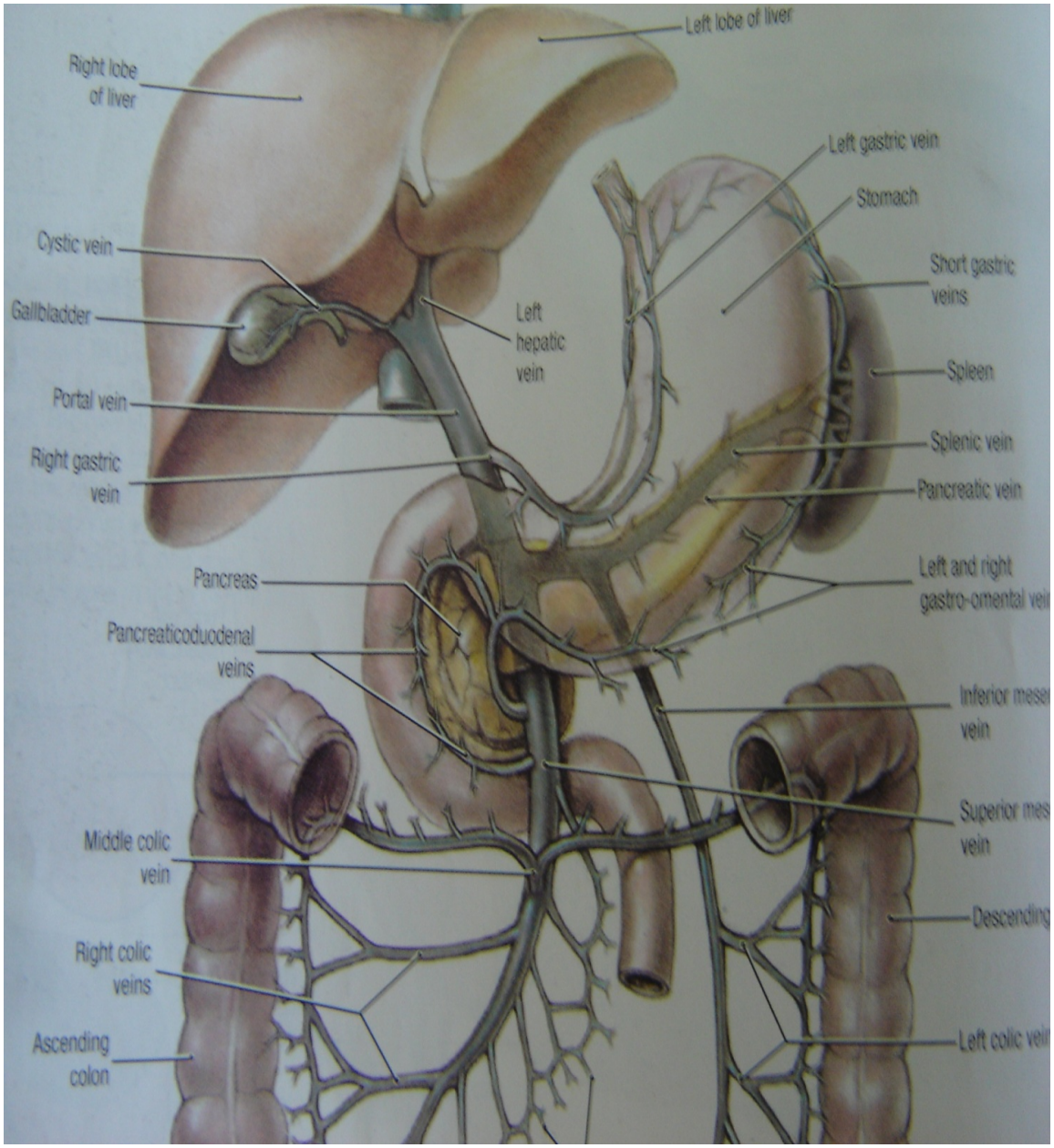
# ANTERIOR VIEW FO THE STOMACH




# NORMAL HISTOLOGY OF THE STOMACH



# ANTERIOR RELATIONS OF THE STOMACH



## PARTS OF THE STOMACH

The main parts of the stomach are the fundus, body and pylorus, with the greater and lesser curvatures forming the left and right borders respectively.

The fundus is the part which project upwards above the level of cardia and is in contact with the dome of the diaphragm. It is usually full of swallowed air.

The largest part of the stomach is body, extending from fundus to the notch, angular incisura, on the lower part of lesser curvature. The lowest border of greater curvature may be above or below umbilical level.

The pyloric part extends from angular notch to gastroduodenal junction and consiste of proximal pyloric antrum which narrows distally as pyloric canal. The circular muscle of distal end of the canal is palpably thickened to form pyloric sphincter. The pyloric sphincter indicated by

1. Pre pyloric veins of Mayo on the anterior aspect. They are two in number, superior and inferior pre pyloric veins.
2. A shallow superficial serosal notch present over the sphincter.

The stomach is completely invested by peritoneum which passes in a double layer from the lesser curvature to liver as lesser omentum and hangs down from greater curvature of the stomach as greater omentum. The lesser omentum that overlies caudate lobe is free of fat and is called as pars flaccida.

## STOMACH BED

Behind the stomach, are a group of structures comprising the stomach bed. The posterior wall is covered by the peritoneum of the anterior wall of the lesser sac. Apart form left crus and some of the diaphragm, the bed consists of the body of pancreas, transverse colon, upper part of left kidney, left suprarenal gland, spleen and left colic flexure and splenic artery. To the right of lesser curvature in the midline lies the aorta with celiac trunk, celiac plexus and ganglia, and celiac lymph nodes

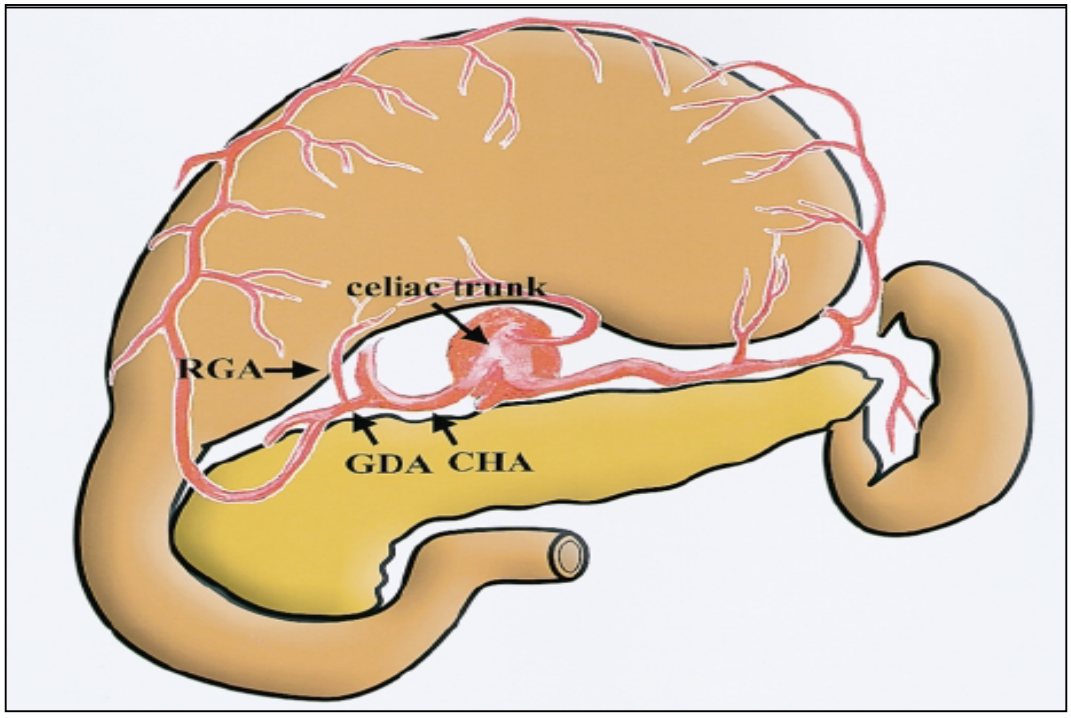
## BLOOD SUPPLY

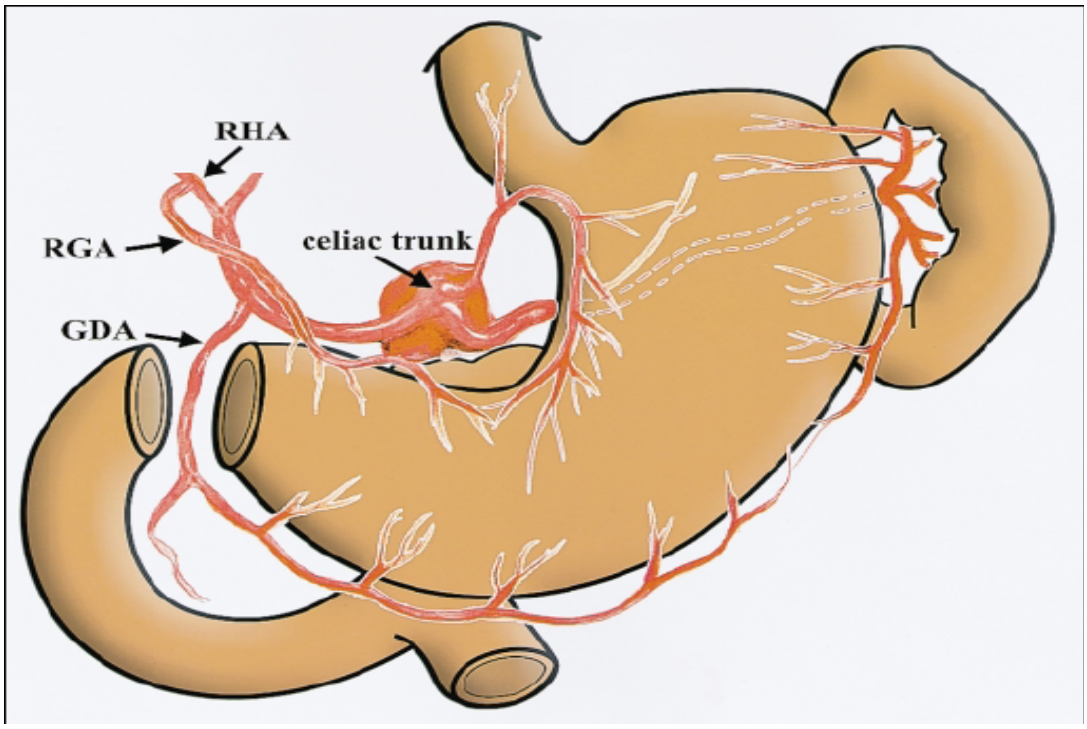
Arterial supply

### 1. Left gastric artery

The smallest branch of the celiac axis runs towards the cardiac orifice and along the lesser curvature form right to left.

# VASCULAR SUPPLY OF THE STOMACH



**1. Right gastric artery**

A branch of the hepatic artery that pursues a course from left to right along the lesser curvature.

**2. The left gastroepiploic artery**

The largest branch of the splenic artery that runs left to right along the greater curvature.

**3. The right gastroepiploic artery**

It arises from gastroduodenl artery and runs right to left along the greater curvature. The right gastroepiploic artery is closer to greater curvature than the left gastroepiploic artery.

**4. Vasa brevia (Short gastric arteries)**

The fundus and left upper part of greater curvature receive about six branches that arise from splenic artery in gastrosplenic ligament. An important feature of gastric circulation is gastric submucous vascular plexus which gives nutritional support to stomach.

Another important branch to be note is **posterior gastric artery.** This is a branch of middle third of splenic artery and runs beneath the posterior peritoneum of lesser omental bursa to fundus of stomach where it enters

through gastrohepatic omentum. In performing gastrectomy, troublesome retroperitoneal haemorrhage occurs if this artery is incidentally damaged

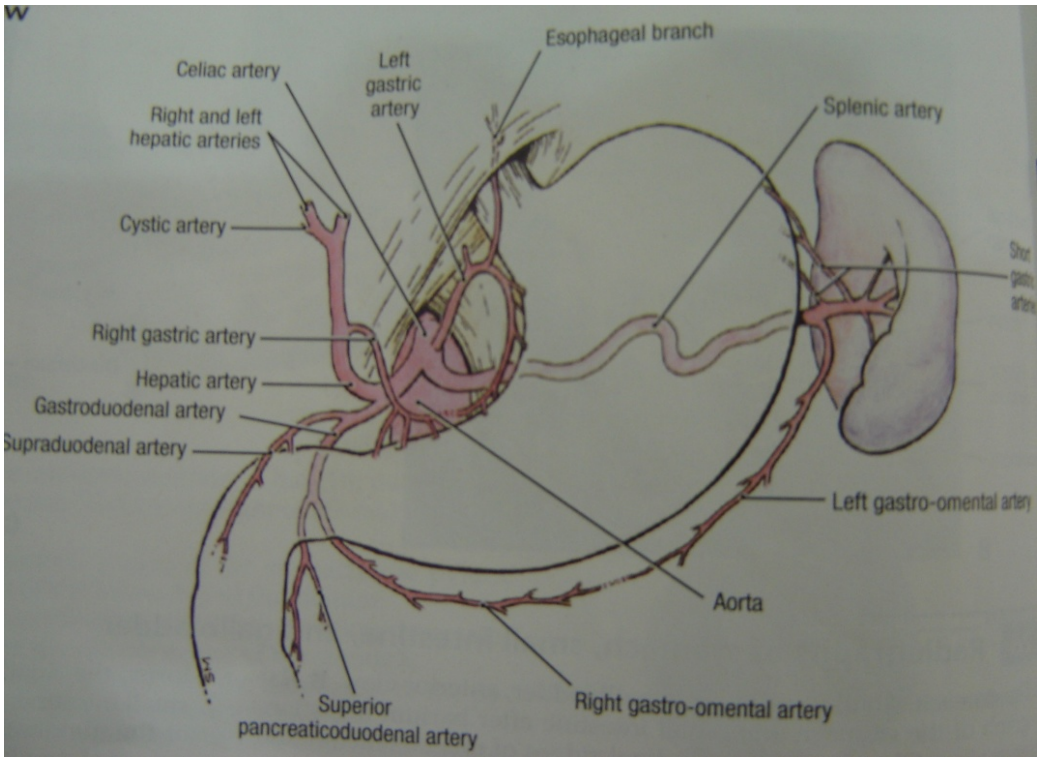
## VENOUS DRAINAGE

Veins of same name accompany the arteries and drain into portal vein itself or to splenic and superior mesenteric vein tributaries. The prepyloric veins (unaccompanied by an artery) drains into the right gastric vein.

## LYMPHATICS

All lymph from the stomach eventually reaches celiac nodes after passing through various outlying groups of lymph vessels which anastomose freely in the stomach wall but there are valves in the vessels that direct lymph in such an way that a line drawn parallel to greater curvature and two thirds of way down the anterior surface indicates a watershed. From largest zone above and to right of this line, lymph passes to left and right gastric nodes. From upper and left quadrant lymph flows to pancreatic splenic nodes at splenic hilum and upper and posterior border of pancreas. From the rest, lymph flows along the epiploic vessels to pyloric nodes. The pyloric part of the stomach drains to hepatic nodes in porta hepatis and pyloric and

# VASCULAR AND LYMPHATIC SUPPLY OF THE STOMACH




left gastric nodes. Rarely left supraclavicular nodes may become involved (Troisier's Sign) by spread along the throracic duct.

## VAGUS NERVE SUPPLY

**'Dragsted'** and his associates introduced the concept of complete division of vagus nerve supply to stomach and found that there was a decrease in the volume and acidity of continuous night secretions in duodenal ulcer patients.

The vagus descends from esophageal vagus plexus in thorax. The vagal trunk forms 3-4 cm above the diaphragem. The left vagus forms a major portion of anterior vagus and right trunk forms the posterior vagus but the two trunks are communicated by several long and short communications.

The anterior trunk is single in only 62% of cases while the posterior trunk is single in 92%. The anterior trunk is found closely applied to the anterior surface of esophagus. The posterior vagus on other hand is separated from esophagus by loose areolar tissue. The anterior trunk gives off 3 branches. The hepatic branch passes within lesser omentum and supplies bile duct, gall bladder and pancreas. The gastric branches are 5 to 10 thin twigs, they penetrate the lesser curvature and travel berwee the serosa and muscle for a distance of1- 2 cm before penetrating the submucosal layer. These

nerves are divided by anterior seromyotomy of the stomach. **The anterior nerve of Laterget or Greater anterior nerve of Mitchell** fans into branches akin to crowfoot that supply anturm and is responsible for **'antral milling'**

Pyloric nerve of McCrea is an inconstant branch. In proximal gastric vagotomy, the gastric branches are only cut, leaving the hepatic and anterior nerve of Laterget intact.

The posterior trunk of vagus divides into 3 branches similar to anterior trunk that instead of hepatic branch, celiac branches are given. It supplies pancreas, small intestine and large intestine till mid transverse colon, the criminal nerve of Grassi is a branch of posterior trunk, that if not cut can cause recurrence of ulcer and symptoms

The hepatic vagal branch of anterior trunk is distributed along greater curvature and forms epiploic nerves. These fibres are also cut in **extended proximal gastric vagotomy**

## APPLIED GASTRIC PHYSIOLOGY

### Regulation Of Gastric Secretion

Gastric secretion is regulated by both nervous and humoral mechanisms. The neurocrine secretion is mediated through parasympathertic fibres of vagus that secrete **acetylcholine.** It increases the secretion by two mechanisms first, it has a direct stimulatory action and second it inhibits somatostatin, which inhibits the gastric secretion.

**Histamine** acts through paracrine route. It is released from mast cells, enterochromaffin cells and nerves.

### GASTRIN

When food enters the stomach, its causes antral portion of stomach mucosa to secrete gastrin. Gastrin stimulates oxyntic cells to secrete hydrochloric acid.

Gastric secretion is inhibited mainly by somatostatin, cholecystokinin and VIP. Secretin which is from ducodenum that inhibits gastric secretion through somatostatin. Neurotensin, substance P and high concentration of alcohol inhibits gastric secretion

## TYPES OF SECRETION

The mucosa of fundus and body contains parietal cells that secrete HCl and intrinsic factor, and chief cells that secrete pepsinogen which is converted to active pepsin in the presence of acid.

## PHASES OF GASTRIC SECRETION

### 1. Cephalic phase

It results from expectation, vision, smell or chewing of food, Neurogenic signals causing the cephalic phase can originate in appetite center in amygdala or hypothalamus. They are transmitted through the vagi of the stomch. This accounts for less than one tenth of gastric secretion.

### 2. Gastric phase

Once food enters into stomach, amino acids present in the food stimulates gastric secretion which in turn promotes secretion of HCL. The presence of food in the stomach produces stimulation via stretch and chemo receptors. The Acetylcholine mediated secretion of gastric juices occur in this phase. The gastric phase accounts for atleast two thirds of total gastric secretion**.**

### 3. Intestinal phase

The presence of carbohydrate, fat and acid that enters the duodenum stimulates the secretion of cholecystokinin and secretin that in turn inhibits the HCL secretion in the stomach, via neural and hormonal mechanisms.

## GASTRIC MOTILITY

The stomach receives and accommodates the ingested food. This adaptive relaxation is mediated through vagal nerves. The loss of adaptive reflex reflects clinically as early satiety. This is seen in patients with vagotomy and also in carcinoma of the stomach patients when the malignancy infiltrated wall does not relax.

Mixing of gastric secretion is caused by weak peristaltic constrictor waves, also called mixing waves that occur every 20 seconds. These waves are initated by basal electric rhythm consisting of electrical slow wave.

The mixture of food with gastric secretions is called chime. The appearance is that of murky semisolid or paste.

Propulsion of gastric secretion occurs when the slow wave is associated with electrical response activity. Each time a peristaltic wave passes over antrum, it digs deeply into contents of antrum, yet the opening of

the pylorus is small that only few milliliters of contents is pushed into ducodenum.

**Emptying of stomach**

It is regulated by signals from both stomach and duodenum.

The stomach signals are mainly twofold

(1) The nervous signals caused by distension of stomach by food and

(2) Release of gastrin

Both of these factors increase pylorus pumping force while slightly inhibit pylorus, thus promoting stomach emptying.

On the other hand, signals from duodenum depress the pyloric pump and increases the pyloric tone. This occurs when

1. Excess volume of chime enters the duodenum or **enterogastric feed back reflexes.**

2. Certain type of chime (increased acidity or more fat contents) enters duodenum, mediated through hormones (CCK, secretin).

## ETIOLOGY

Gastric outlet obstruction is on obstruction occurring at the level of pylorus or duodenum due to various causes. The term pyloric stenosis is normally a misnomer. The stenosis is seldom at the pylorus. It the obstruction is due to peptic ulcer disease. It is mostly due to stenosis found in the first part of duodenum. True pyloric stenosis is ulcer disease, occurring due to pyloric channel ulcers.

The two common causes of gastric outlet obstruction are:

1. Gastric malignancy and
2. Pyloric stenosis secondary to peptic ulcer disease

Previously the cicatrizing duodenal ulcer was the commonest cause of gastric outlet obstruction. Now with the wider usage of the endoscopy and potent anti- ulcer medical treatment decrease in the incidence of peptic ulcers, gastric outlet obstruction should be considered malignant until proven otherwise. Previously the incidence of gastric outlet obstruction in the ulcer patients were high up to 8.5 to 10%. But with the recent anti- uncer drugs; this complication of gastric outlet of obstruction due to ulcer has reduced to about 2.5%.

## OTHER CAUSES OF GASTRIC OUTLET OBSTRUCTION

1. Other malignancies

   Gastric lymphomas

   Pancreatic carcinoma at the head

2. Strictures - by acid ingestion

   - By alkali ingestion

3. Benign adenomatous polyp

4. Inflammations

   a. Cholecystitis

   b. Acute pancreatitis

   c. Crohn's disease

   d. Eosinophilic gastroenteritis

   e. Gastro intestinal tuberculosis

5. Miscellaneous causes

   a. Adult hypertropic pyloric stenosis

   b. Post surgical stenosis

   c. Pyloric, antral and duodenal webs / diaphragem

   d. Duodenal bands/ megaduodenum

   e. Submucous Lipoma – antral area.

   f. Annular / ectopic pancreas

g. Down's syndrome

h. Behcet' disease

i. Superior mesenteric artery compression syndrome or wilkie's syndrome OR cast syndrome

j. Bezoars

k. Choledochal cyst/ pseudocyst

l. Post operative adhesions

m. Bouveret's syndrome (due to gallstone obstruction in duodenum).

## ADULT HYPEROPHIC PYLORIC STENOSIS

As in infantile stenosis, the pylorus is enlarged and the circular muscle fiberes show uniform hyperplasia and loss of peptide immunoreactivity in nerve fibres. Occasionally presentation is delayed until adult life but some of the cases reported may be secondary to peptic ulcer disease. Families are reported with both adult and infantile forms of the disease. Barium meal shows a narrow elongated pyloric channel, which can be mimicked by a carcinoma. Because of this possibility, the narrow area in adult hypertrophic stenosis in usually resected in preference to pyoloromyotomy.

## ANTRAL WEB (PRE PYLORIC MEMBRANE)

This condition may present as a congenital failure of recanalization while it nearly always present in neonates. Cases have been reported in adults with intermittent pyloric obstruction and although these are probably secondary to gastric ulceration. Barium examination shows a Double Bulb effect' one bulb being the normal duodenal bulb and other the region between membrane and pylorus. Surgical excision of the membrane is indicated.

**SUPERIOR MESENTRIC ARTERY SYNDROME**

The third part of duodenum is compressed and obstructed by superior mesenteric artery. This is also called as vascular compression of the duodenum OR arterio mesenteric duodenal compression OR Wilkie's disease or cast syndrome. The third part of duodenum is compressed between the mesenteric artery or sometimes by one of its two branches in transverse mesocolon, and lumber vertebra, prevertebral muscles and aorta.

In normal person, mass of fat and lymphatic tissue around the origin of superior mesenteric artery protects from compression. So the precipitating factors include weight loss, rapid growth in height, and increased lordosis, a short mesentery, high attachment of ligament of Treitz or this patients.

The symptoms of chronic vascular compression is relieved by posture. Barium meal series often confirms the diagnosis. Hypotonic duodenography is superior to barium meal series.

**BEZOARS**

Bezoars are concretions of foreign materials found in stomach and intestines. They are classified as trichobezoars (hair balls) phytobezoars and other concretions (made up of various ingested chemical substances).

Obstruction is the most common compilation. Small bezoars composed mainly of food substances can be fragmented through fibre optic endoscope; the fragments then will pass spontaneously through the bowel. Laser based endoscopic lithotripsy device has been used to break up gastric bezoars.

Large bezoars, particularly hairyballs may not be amenable to any treatment other than open surgical removal by means of gastrotomy.

## BOUVERETT SUNDROME

Rarely a large gall stone can pass all along the common bile duct and obstruct the duodenum resulting in gastric outlet obstruction.

## ANNULAR / ECTOPIC PANCREAS

This is a result of complete rotation of the ventral pancreatic bud during development, so that a ring of pancreatic tissue surrounds the second or third part of the duodenum. Plain x-ray shows double bubble sign, interrupted gastroduodenal shadow produced by rigid ring of pancreatic tissue. The usual treatment is bypass (duodenojejunostomy or duodenoduodenostomy. Attempts at resection of band may result in pancreatic fistula.

Ectopic pancreas can be found in submuucosa in parts of the stomach, duodenum, small intestine or liver. Ectopic pancreas in the wall of the intestine may give rise to obstruction if inflamed.

**1. FROM THE HISTORY**

a. Abdominal Pain

- ❖ Nature
- ❖ Duration
- ❖ Aggravating factors
- ❖ Relieved by vomiting

**b. Vomiting**

a. Projectile in nature

b. Usually nocturnal; at the end of day,

c. Pain abdomen pain tends to be more constant than previously

d. Pain relieved by vomiting

e. It is of large volume

f. Free of bile

g. Often containing food recognized as having been caten 12 to 24 hours before.

**2. Physical Examination**

- Visible gastric peristalsis (VGP)
- Succussion splash more than 4 hours after eating

**3. Gastric aspirate**

- Gastric residual volume greater than 300ml 4 hours after eating

**4. Radiography**

- Large atonic stomach, with retained food
- Retention of barium in the stomach more than 50% at 4 hours.

**5. Special studies**

**(i) Saline Load Test**

Definite gastric outlet obstruction present when more than 400ml of 750 ml of 0.9% NaCl is retained in the stomach after 30 minutes.

**(ii) External scanning technique**

Done by using Technitium istope

Liquid phase: fractional emptying rate of less than 10% per minute.

Solid phase: fractional emptying rate of less than 0.5% per minute.

## ETIOPATHOLOGY AND CLINICAL FEATURES

In peptic ulcer disease, narrowing of the lumen in the region of pylorus may result from spasm, acute inflammation and edema, muscle hypertrophy or contraction of scar tissue. Of these, rapidly reversible elements include spasm, edema, inflammation and pyloric dysmotility related to ulcer and inflammatory change. Fibrosis, scarring and deformities are the irreversible

cause of obstruction. Gastric atony developing after prolonged obstruction, contributes to gastric retention.

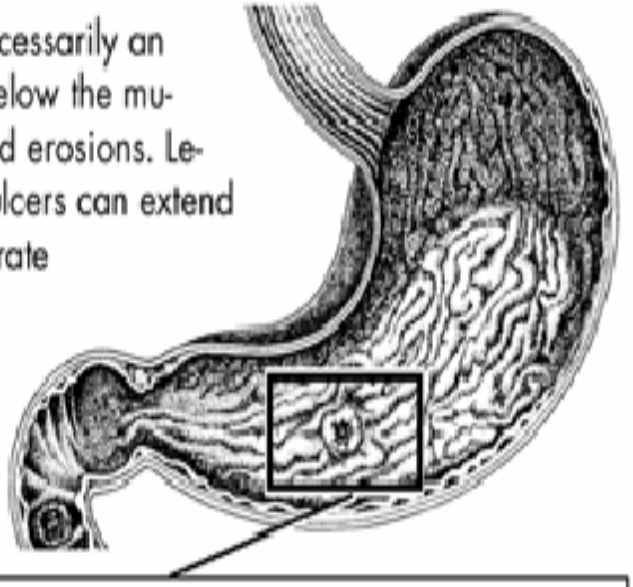
**SYMPTOMS AND SIGNS**

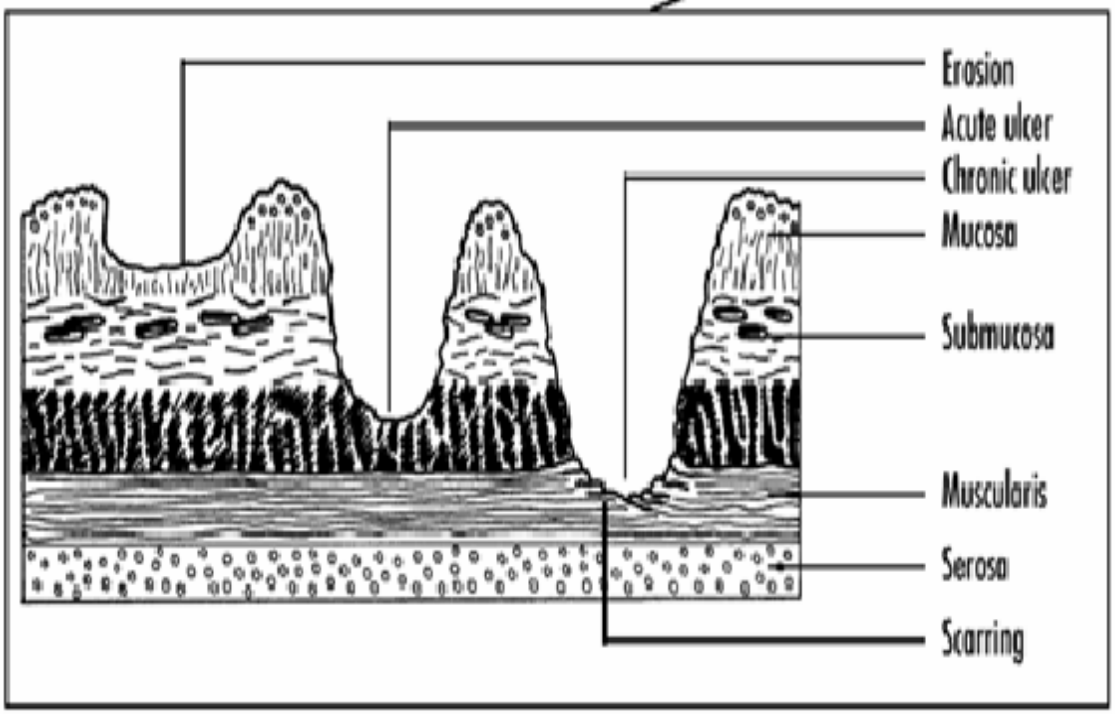
In gastric outlet obstruction due to ulcer a history of antecedent ulcer symptoms may be elicited. With the onset of gastric stasis, a change in the rhythm and character of distress often occurs. The previously well localized, gnawing discomfort is supplanted by a more vague diffuse sense of epigastric fullness. The pain may be due to vigorous peristalsis as the stomach attempts to overcome the obstruction. The pain may reduce with vomiting. As the degree of obstruction increases, the stomach further dilates and the vigor of peristalsis declines. This is the state of gastric decompensation. In this stage vomiting is less frequent but may be more copious.

The normal or excessive appetite often associated with an uncomplicated ulcer is succeeded by anorexia leading to weight loss. Constipation is a rule but diarrhea occurs accasionally.

In about two third of patients, a distinct succusion splash is obtained in fasting state. If the stomach musculature has not lost its tone, visible

A gastrointestinal lesion is not necessarily an ulcer. Lesions that don't extend below the mucosal lining (epithelium) are called erosions. Lesions of both acute and chronic ulcers can extend through the epithelium and perforate the stomach wall. Chronic ulcers also have scar tissue at the base.





peristalsis maybe observed traversing the upper abdomen from left to right side.

In gastric outlet obstruction due to carcinoma stomach, the patients are usually elderly and the history will be short, there will be a history of loss of appetite and loss of weight, episodes of hematemesis or malena may be present. Anaemia or jaundice with cachaxia may be present. Hard nodular lump may be present in relation to the pylorus in 10-15% cases.

The stomach is less dilated as compared to pyloric obstruction in duodenal ulcer, in gastric outlet obstruction due to other causes, the clinical features present accordingly.

**METABOLIC DISTURBANCES**

Prolonged vomiting of gastric contents results in a characteristic series of electrolyte disturbances. Initially the major loss in fluid rich in hydrogen ($H^+$) and chloride $Cl^-$ ions so that a minor degree of dehydration may accompany a hypochloremci alkalosis. At this stage, the serum sodium is usually normal and hypokalemia may not be obvious.

The more marked metabolic change which accompany unrelieved obstruction result form a combination of continued losses with a secondary

change in renal perfusion. The excretion of an alkaline urine entails further loss of sodium and plasma volums. This in turn result in reduced gloumerular filtration rate. Sodium is ultimately reabsorbed from the tubules in exchange for potassium and hydrogen ions and the urine then becomes acid the so called paradoxical aciduria of stenosis. Hence the alkalosis becomes more severe and hypokalemia become more marked.

As the secondary effect of the alkalosis, the concentration of plasma ionized calcium may fall so that disturbances of conscious level and tetany may be apparent.

## INVESTIGATIVE MODALITIES

The single most reliable clinical test for gastric retention is aspiration of stomach by a tube of an abnormally large volume of gastric contets. If the history and further clinical examination suggest gastric stasis, immediate aspirationis a simple maneuver and is oftern helpful in establishing the diagnosis, in order to withdraw, retained food particles, a large caliber tube may be needed.

## SALINE LOAD TEST (SLT)

A method to test the capacity of stomach to empty itself of a test meal was described by "**Goldstein and Boyle".** To summarize, the procedure begins with the placement of NO.16 French Nasogastric sump tube into the stomach. Position the tip 65 cm form the nares and empty the stomach. Place the patient right side down to enhance gastric emptying. Infuse 750 ml of normal saline (0.9% NaCL) in to the tube for over three of five minutes. After thirty minutes use a syringe to aspirate and measure gastric residual volume. Interpretation of gastric residual volume is as follows.

**SOLID MEALS**

Meals consisting of various food substances have been advocated to assess gastric emptying. One of these the **Barium Burger Test** was compared with saline load test and both were compared with conventional barium meal. The result indicated that a meal of food rather than fluid provides a more exacting test of stomach ability to empty itself. Mild gastric retention was detected by barium burger test, when the saline load test and conventional barium radiographs were normal. The barium burger test, in addition to being a more sensitive index of gastric emptying further offers the advantage over the saline load test of not requiring intubations or special equipment, other than the ordinary radiographic apparatus.

## SCINTIGRAPHY

Isotope labelling provides a more elegant methods for determining the rate of gastric emptying. It is the most accurate method of detecting the outlet obstruction. Isotope analysis, however, demands equipment not widely available as radiographic equipment and is more expensive and requires constant monitoring.

## RADIOLOGY

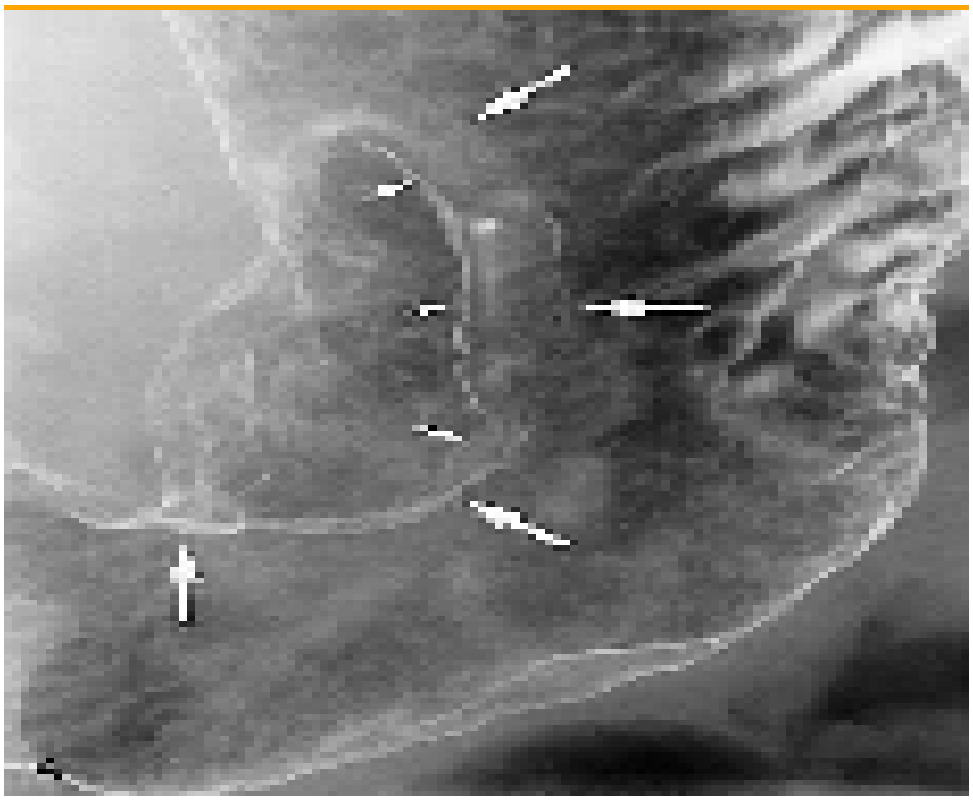
A plain film of the abdomen, preferably taken with the patient upright, often shows the shadowy outline of a distended stomach. A gas bubble can be identified within the stomach by a characteristically “fuzzy” fluid level described by Caruso and Berk. This is distinct from sharp, even air fluid levels in other segments of the gut, in abscess cavity or in hydropneumoperitoneum.

An excessive overnight gastric residue is easily detected by alert fluoroscopist after the patient has taken only one or two swallows of barium. If a large residue is detected, it is usually of little value to proceed further until the stomach contents have been completely aspirated. In the radiographs, the admixture of barium and even modest amount of retained

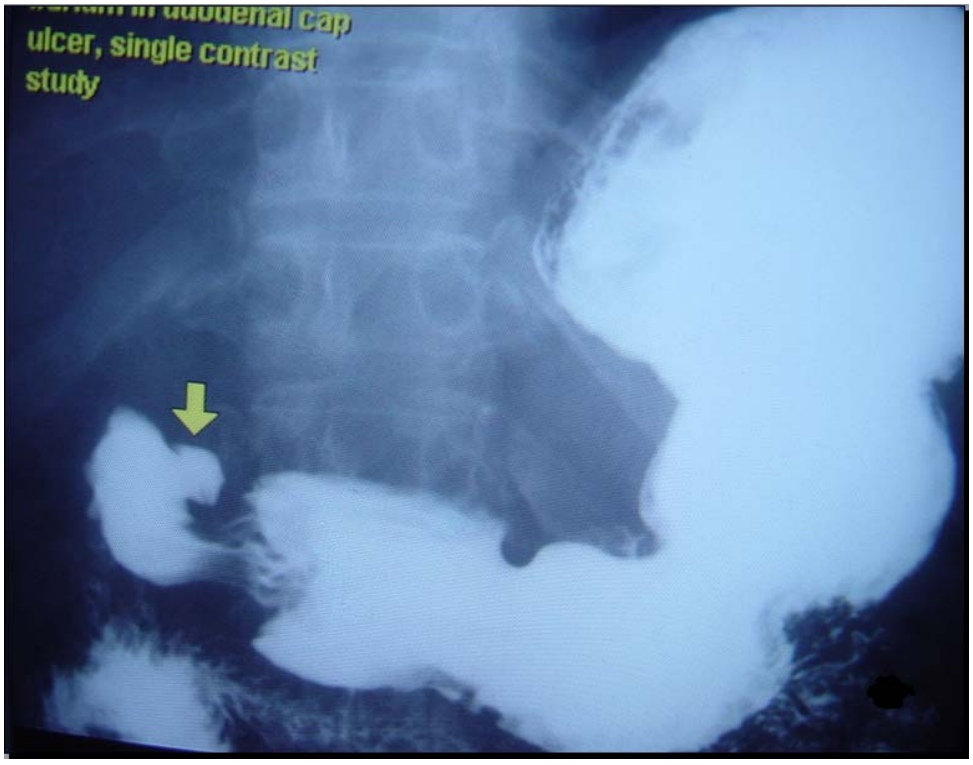
non opaque residue can be identified by its mottled density. The greater the dilation of the stomach, the greater is the possibility of organic stenosis. The most distended stomach is produced in slowly developing pyloric stenosis from peptic ulcer of duodenum.

Of significance radiographically is the length of time that barium is retained in the stomach. The bulk of the barium meal is evacuated by normal stomach in two to four hours and normal stomach should not contain barium after 6 hours. If moderate retention is observed in instances in which relatively active peristalsis is seen fluoroscopically, the peristalsis from extra gastric disease or the effect of opiate or anticholinergic drugs or vagotomy must be considered. In cases of almost complete pyloric obstruction barium may be retained in stomach for 24 hrs or longer.

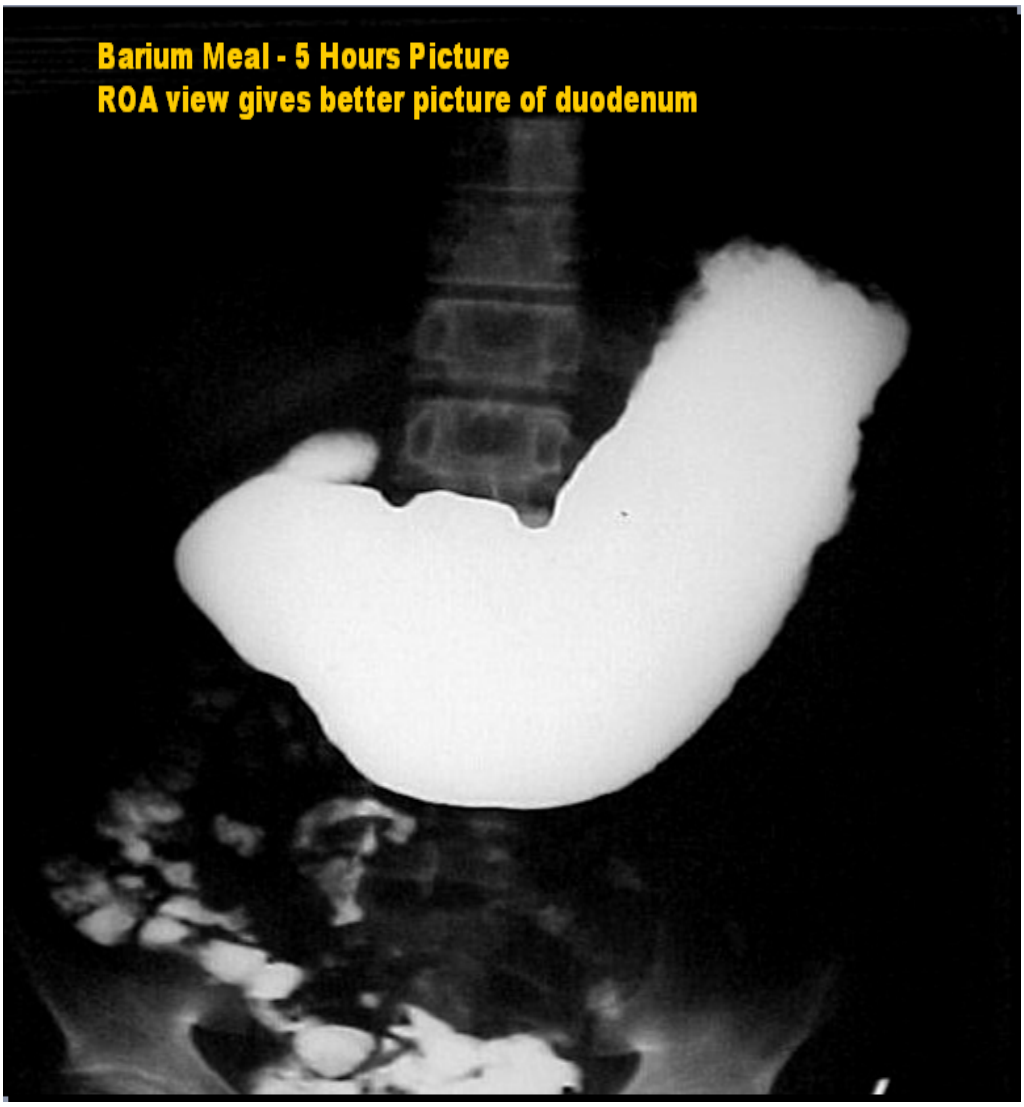
**BARIUM STUDY- SHOWING GASTRIC OUTLET OBSTRUCTION**



**BARIUM STUDY DEFORMED DUODENAL BULB**



# BARIUM FILM SHOWING GASTRIC OUTLET OBSTRUCTION



**ENDOSCOPY SHOWING TUMOUR CAUSING GASTRIC OUTLET OBSTRUCTION**

**COMPLETE GASTRIC OUTLET OBSTRUCTION BY THE TUMOUR**

## ENDOSCOPY

Fibreoptic endoscopy now is an essential tool in diagnosis of the outlet obstruction in defining the obstructive lesion. It can differentiate malignancy from chronic duodenal ulcer. Moreover multiple mucosal biopsies may be obtained. Even when apparently normal intact mucosa covers the unyielding gastric outlet, a small brush can be inserted through the stenotic pylorus to obtain material for cytologic examination (Brush Cytology)

Detecting the mucosal irregularities usually associated with early gastritis like carcinoma can be enhanced by endoscopic dye spraying with vital dyes, such as **0.1% indigocalmin.**

## ENDOSCOPIC ULTRASONOGRAPHY (EUS)

Endoscopic ultrasonography uses a high frequency (7.5 or 12 MHz) transducer at the end of an endoscope and allows highly accurate staging of the malignancy an perigastric lymphnodal status.

CEA is elevated in approximate 1/3 of cases of carcinoma of the stomach, but sensitivity is low. Elevated β HCG and CA 125 in gastric cancer before chemotherapy may reflect not just tumour burden but aggressive biology.

## POSITRON EMISSION TOMOGRAPHY

This investigation is done by using ($^{18}F$) flourodeoxy glucose (FDG). Positron emission tomography has a sensitivity ranging from 92% to 100%, accuracy of 90% to 96% and specificity of 100%. But still it is in the experimental stage.

## LAPAROSCOPY

Laparoscopy has an accuracy of 94%. The invent of laparoscopic ultransonographic probes added a third dimension to the laparoscopic examination. Whenever feasible we should elminiate the need for patients undergoing laparotomy without resection in carcinoma of the stomach.

## GASTRIN LEVEL

Hypergastrinemia has been associated with outlet obstruction. This is due to gastric antral distension due to retained food. If the gastrin level persists even after surgery, we need to rule out Helicobacter pylori infection and gastrinomas.

## MANAGEMENT

Medical Management

When manifesting in a surgical setting, obstruction is often accompanied by severe, long standing ulcer disease. Initial steps in management are to confirm the diagnosis of gastric retention and then to empty and decompress the stomach. If gastric retention is confirmed, a large bore **Ewald's tube** is put stomach lavaged to remove debris. Then the small sized nasogastric tube should be replaced and intermittent suction continued for 3 to 5 days to decompress the stomach while intravenous fluid and electrolytes are administered. In some cases prerenal azotemia, hyponatremia and a hypokalemic metabolic alkalosis occurs due to severity. These cases are treated with intravenous normal saline with potassium supplements.

After the patient is stabilized, repeating the saline load test can gauge the progression, residual volume less than 200 ml indicates resolution. Definitive diagnosis by endoscopy is delayed until this initial 3 to 5 days of decompression. About half of the obstructed cases due to ulcer disease respond to this regimen. Management is simplified in these cases due to spasm, edema inflammation or associated dysmotility rather than malignancy or scarring.

Identifying and treating underlying cause is next step. Helicobacter pylori with gastric outlet obstruction will respond to medical therapy over time after cure of H.pylori infection. Similarly gastric outlet obstruction due to NSAID induced ulcer are treated with medical management and avoidance

of NSAIDs. The combination of curing H.pylori infection and continued antisecretary therapy, will eventully reverse duodenal antral deformity

**ENDOSCOPIC BALLOON DILATION**

The normal pyloric channel measuring 10 to 20 mm in diameter, dilates upto 25 mm without difficulty. The inability to pass a standard endoscope with a diameter of 11 mm is suggestive of pyloric stenosis. A pyloric ring less than 5 mm is associated with symptoms.

A Balloon with an inflatable diameter of 10 to 15 mm is used initially. Balloons of 6 to 8 cm in length are preferred because shorter balloons are difficult to maintain in position. The balloon is inflated to maximal pressure with water or diluted contrast medium if fluoroscopy is used.

Dilation is repeated three or four times, each time balloon maintained at maximal inflation for at least one minute.An alternative procedure is dilation over a wire under fluoroscopic guidance. The optimal diameter to be achieved during the dilation is supposed to be 12 to 14 mm and there may be and advantage of postponing the dilation beyond 15 mm until after a period of time and medical management.

With the definite risk of perforation, patient should be appropriately prepared for surgery before dilation and monitored closely after a period of time and medical management.

A post procedure gastrograffin study will generally detect perforation in a timely fashion.

**SURGICAL MANAGEMENT**

Gastric outlet obstruction due to chronic duodenal ulcer

The results of surgical treatment, in which the proper operation has been chosen for the carefully selected patients, are generally, quite satisfactory and ensure against recurrences of the disease.

Vagotomy with antrectomy is the operation of choice if duodenum can be closed securely. Unfortunately in most of the cases duodenum is scarred and closure may not be possible. Truncal vagotomy with posterior vertical retrocolic isoperistaltic no (short) loop, gastrojejunostomy is an easy procedure that is done in scarred duodenum. Truncal vagotomy with retrocolic gastro jejunostomy is preferred because of fewer complications like poor gastric emptying afferent loop syndrome, volvulus, etc.

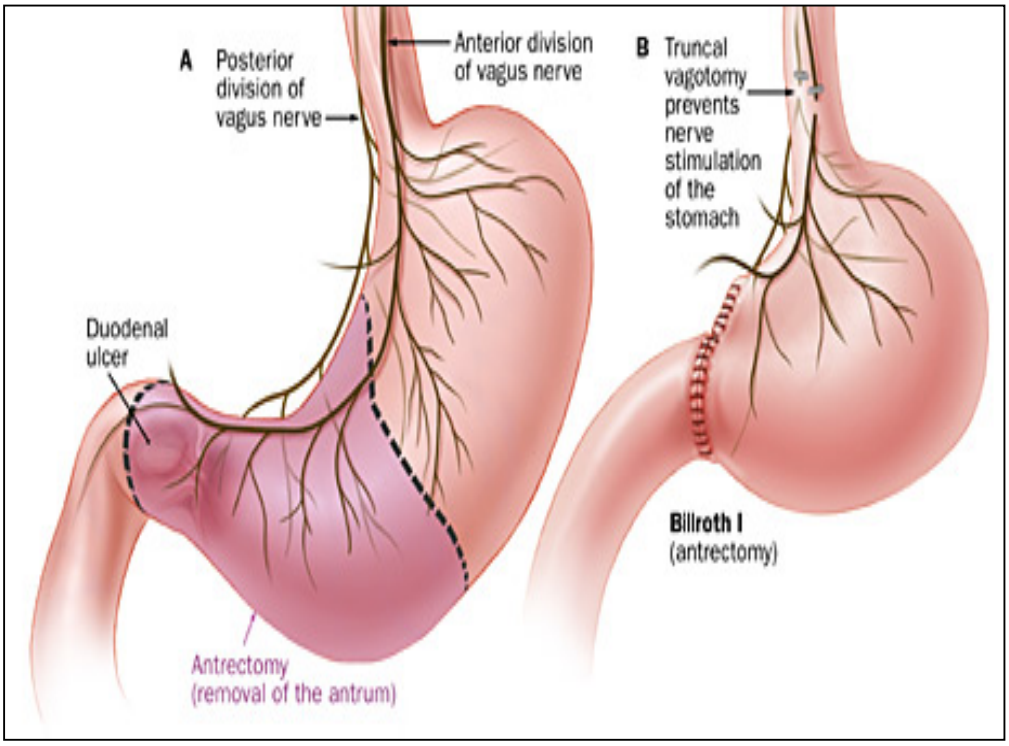
Pyloroplastry through stricured duodenum in often not possible. In such cases, vagotomy with finney's pyloroplastry or vagotomy with jaboulay

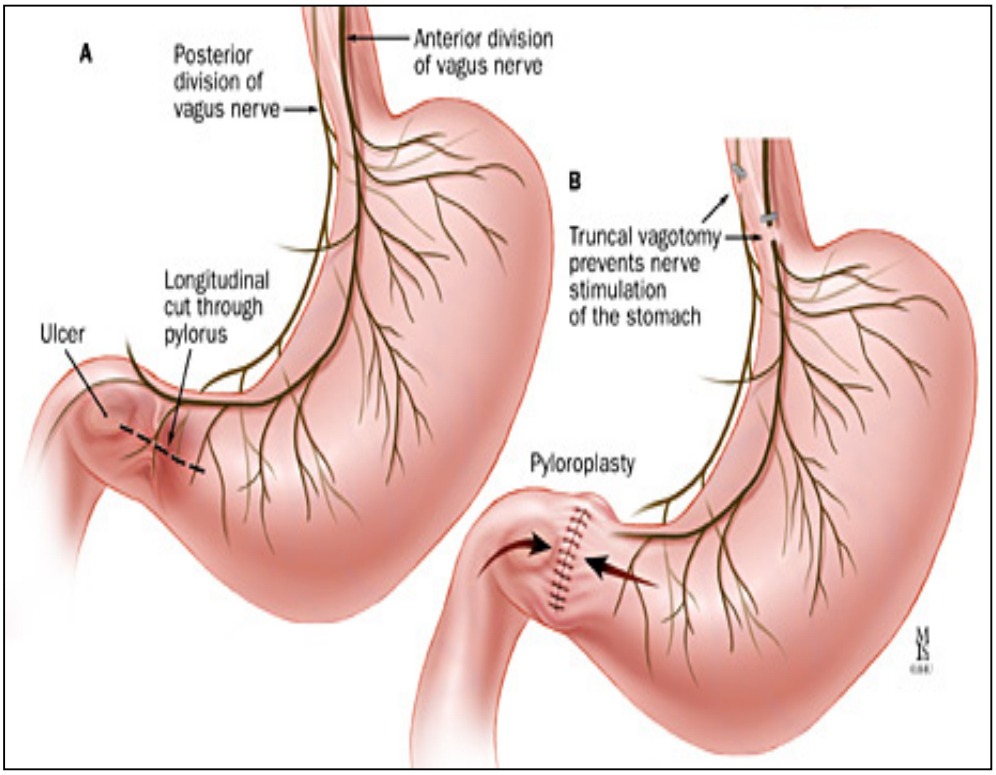
gastroduodenostomy are necessary to bypass the obstructed segment. Highly selective vagotomy with dilation of duodenal scar has been reported with a high recurrence rate due to gastric stasis.

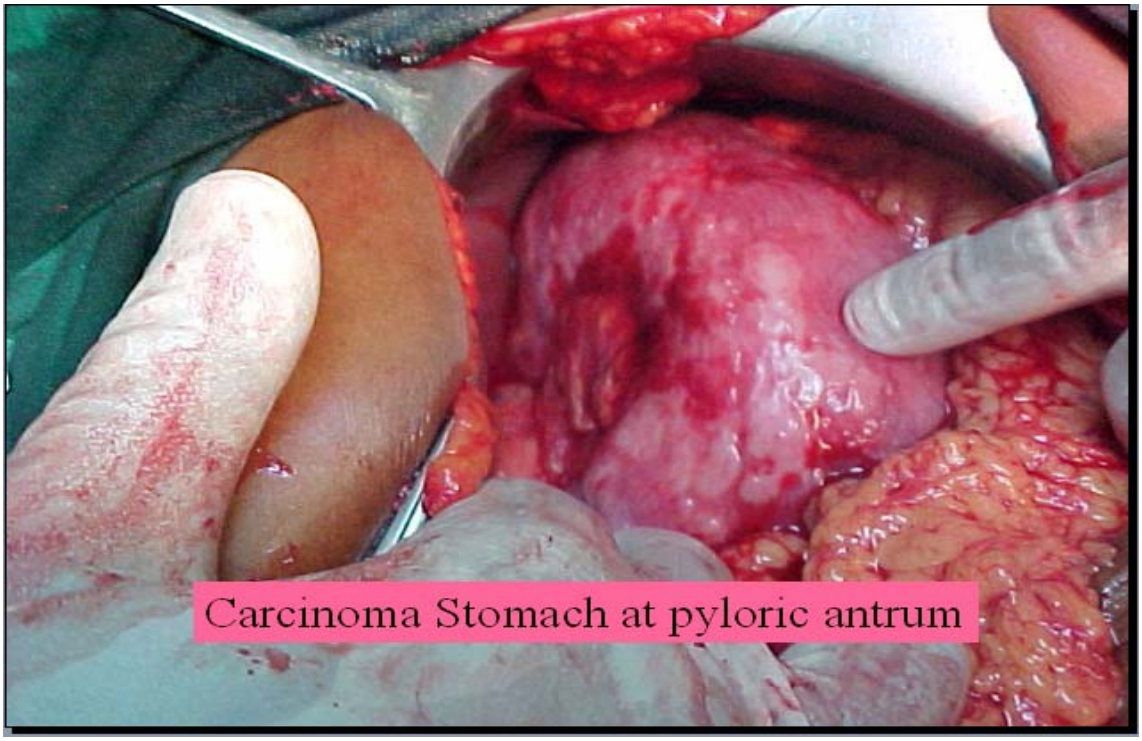
The major post operative problem following uncomplicated surgery for obstruction is delayed gastric emptying. Adequate gastric emptying will be restored in most patients within 5 to 10 days. However in a few patient particularly in those with long standing pyloric obstruction delayed gastric emptying could last for weeks and rarely for months, hence advisability of placing gastrostomy and a feeding jejunostomy. If delayed gastric emptying persists beyond 10 to 14 days a Gastrograffin swallow is obtained to rule out a mechanical obstruction.

After 3 weeks, a flexible gastroscopy may be performed safely to examine the anastomosis. In most instances the problem is gastric atony and not mechanical anastomtic obstruction. The cause of the prolonged gastric

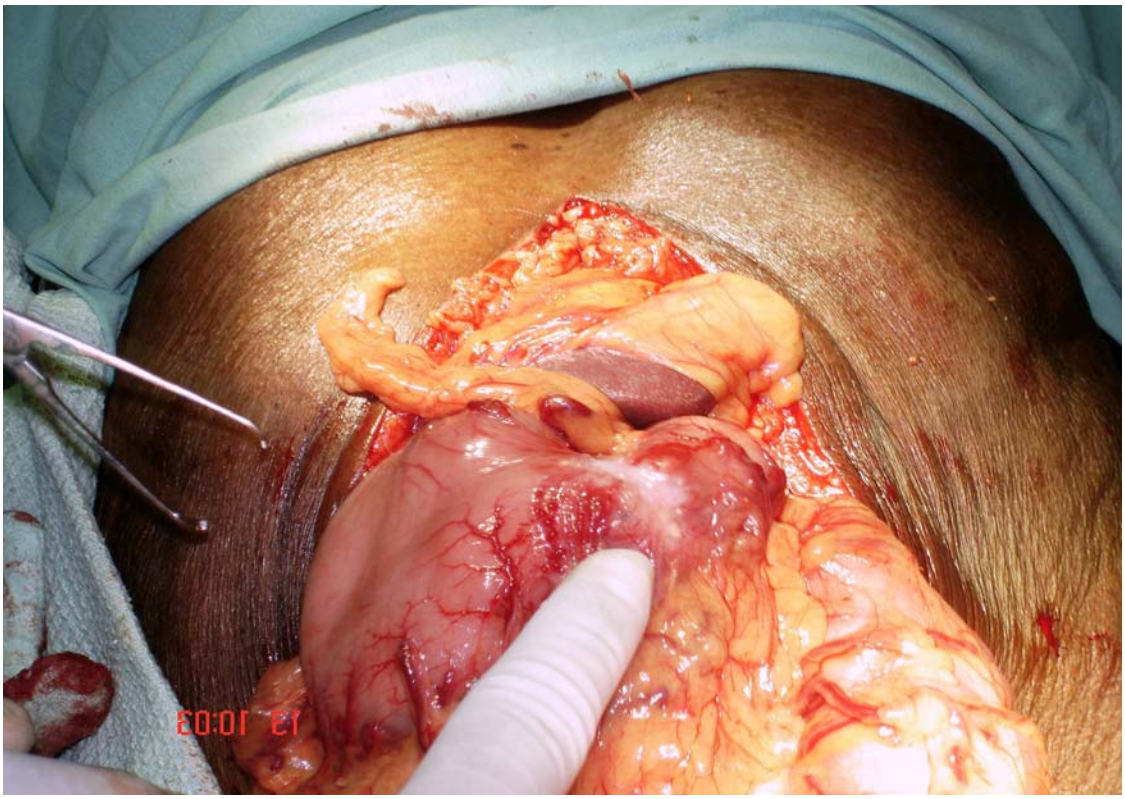
**BILLROTH I GASTRECTOMY**


A
Posterior division of vagus nerve
Anterior division of vagus nerve
B
Truncal vagotomy prevents nerve stimulation of the stomach
Duodenal ulcer
Billroth I (antrectomy)
Antrectomy (removal of the antrum)

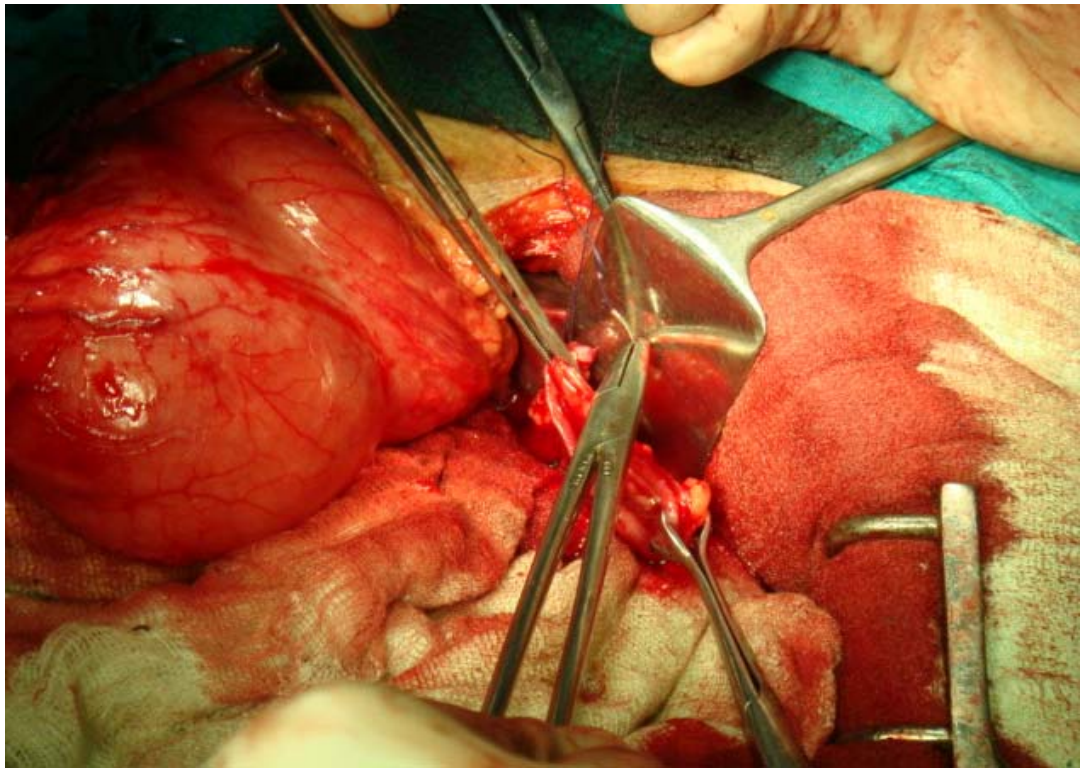

A
Posterior division of vagus nerve
Anterior division of vagus nerve
B
Truncal vagotomy prevents nerve stimulation of the stomach
Longitudinal cut through pylorus
Ulcer
Pyloroplasty



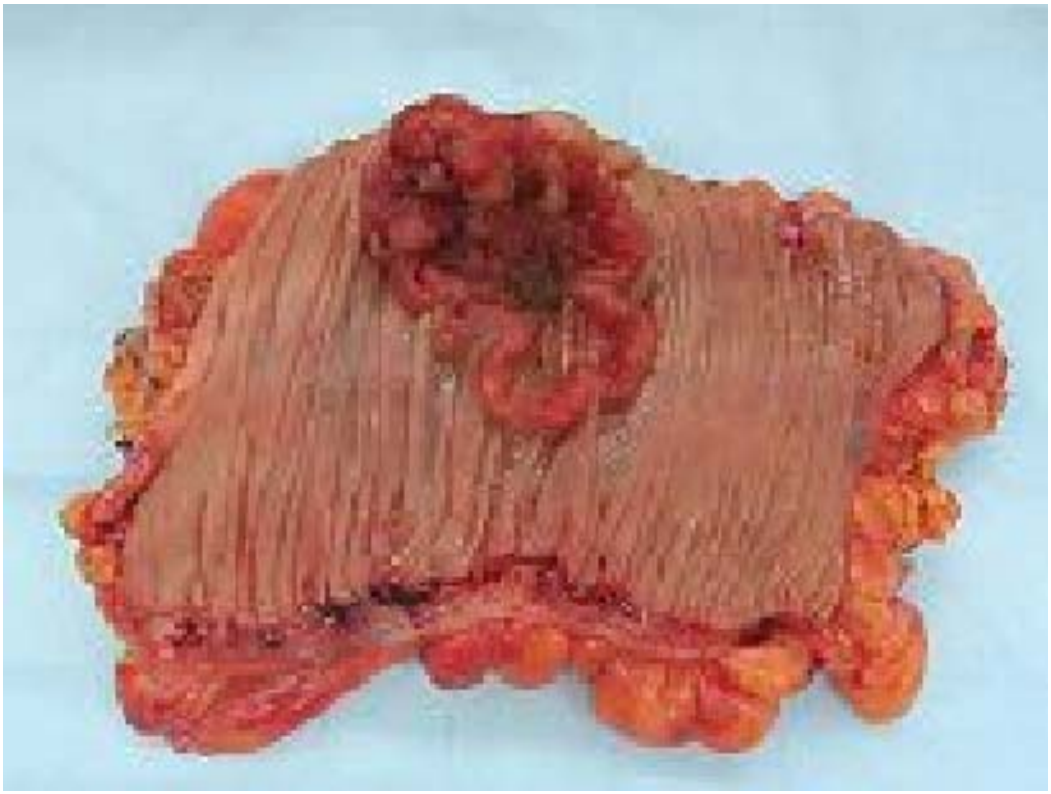

**CARCINOMA STOMACH AT PYLORIC ANTRUM**



# DUODENAL STUMP CLOSURE



**RESECTED SPECIMEN OF CARCINOMA STOMACH**



ileus is uncertain, but may be related to intramural edema from prolonged obstruction.

Prokinetic agents are notoriously unsuccessful in Improving emptying. During the entire time of poor gastric emptying adequate nutrition is maintained in a jejunostomy. The use of omeprazole per jejunostomy significantly reduce gastric secretion and gastrostomy tube losses. If gastric secretion is excessive, the gastrostomy fluid can be rerurned into the intestine via jejunostomy.

The other complications associated with ulcer surgeries are bile vomiting, early and late dumping syndromes (post cibal syndrome), post vagotomy diarrhea, gallstones and stump carcinoma (usully a lag period of more than 10 years).

**GASTRIC OUTLET OBSTRUCTION DUE TO CARCINOMA OF ANTRUM**

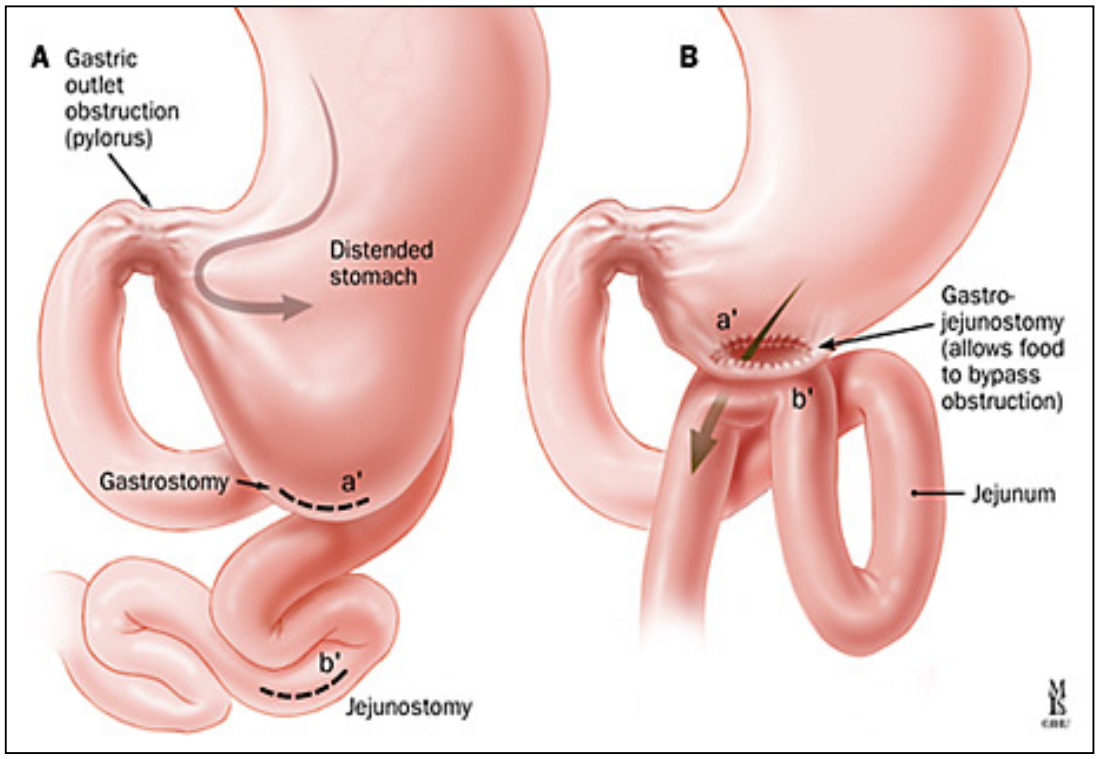
An adequate surgical resection remains the only effective treatment which offers a chance of cure or long survival. Further more,

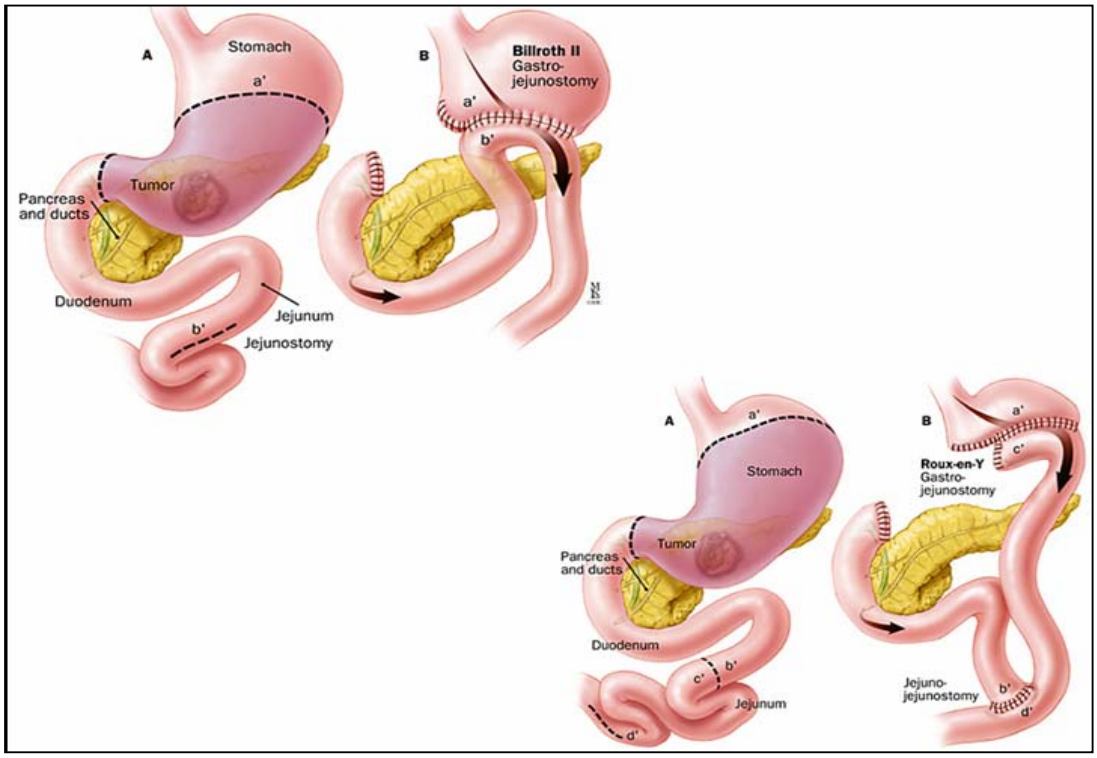
A palliative resection whenever feasible is more effective in relieving symptom than bypass or intubation procedures.

The procedure of choice is **subtotal gastrectomy'** followed by **Billroth II Anastomosis**. This surgery for distal growth of the stomach involves separation of greater omentum from the transverse colon in its entirety, separation of lesser omentum from liver, freeing the perigastric lymphnodes and together with fibrofatty in which they lie.

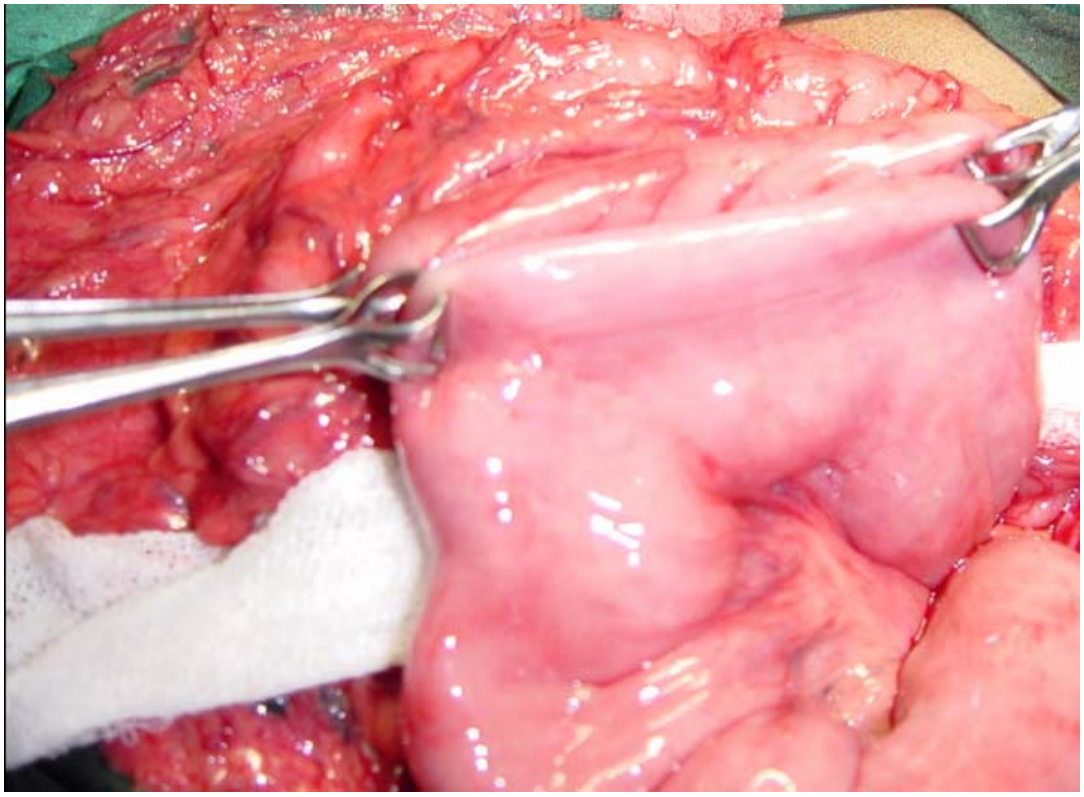
The left gastric and right gastro epiploic vessels, are divided at their origin, stripping all fatty and lymphatic tissue around them toward origin, stripping all fatty and lymphatic tissue around them towards the stomach. For inoperable cases long loop (for about twenty to thirty centimeter) gastrojejunostomy is performed well away from the tumour, so that the advancing growth will not invade the anastomosis. An antecolic anastomosis is preferred because the mesocolon may already be or in future become invaded with tumour at which time patient may have obstruction. A long loop GJ is done, for is gives adequate space for the transverse colon to distend.

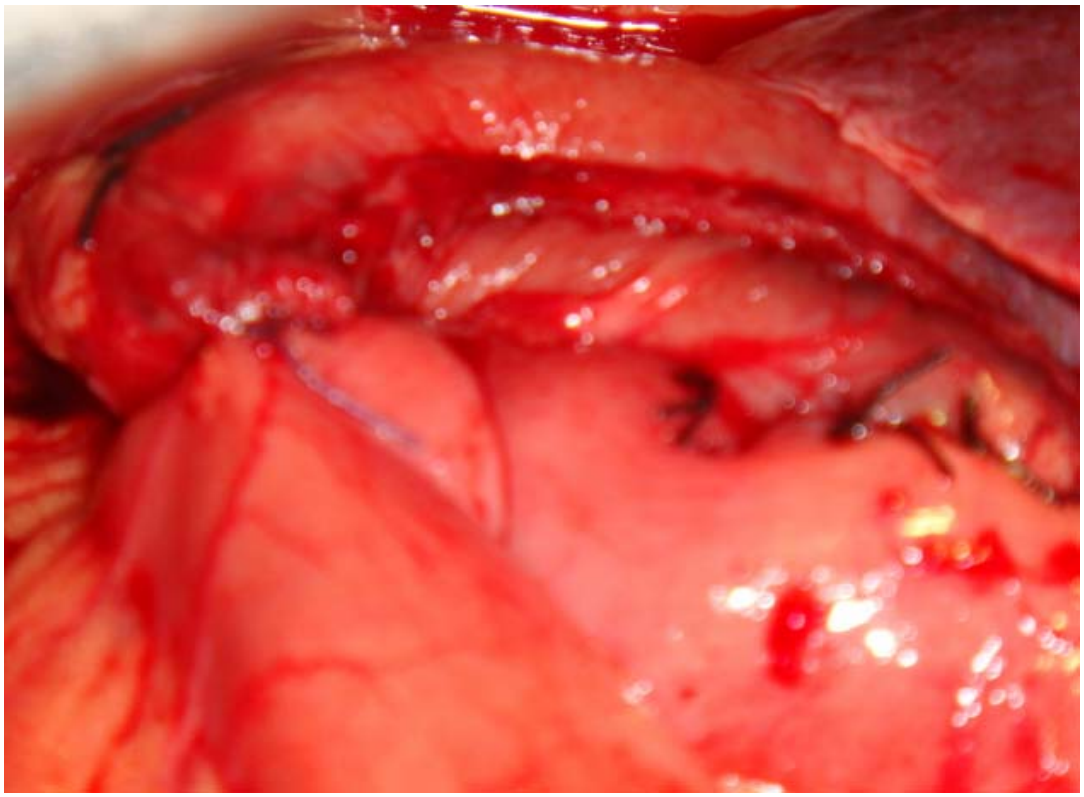
# BILLROTH II GASTRECTOMY



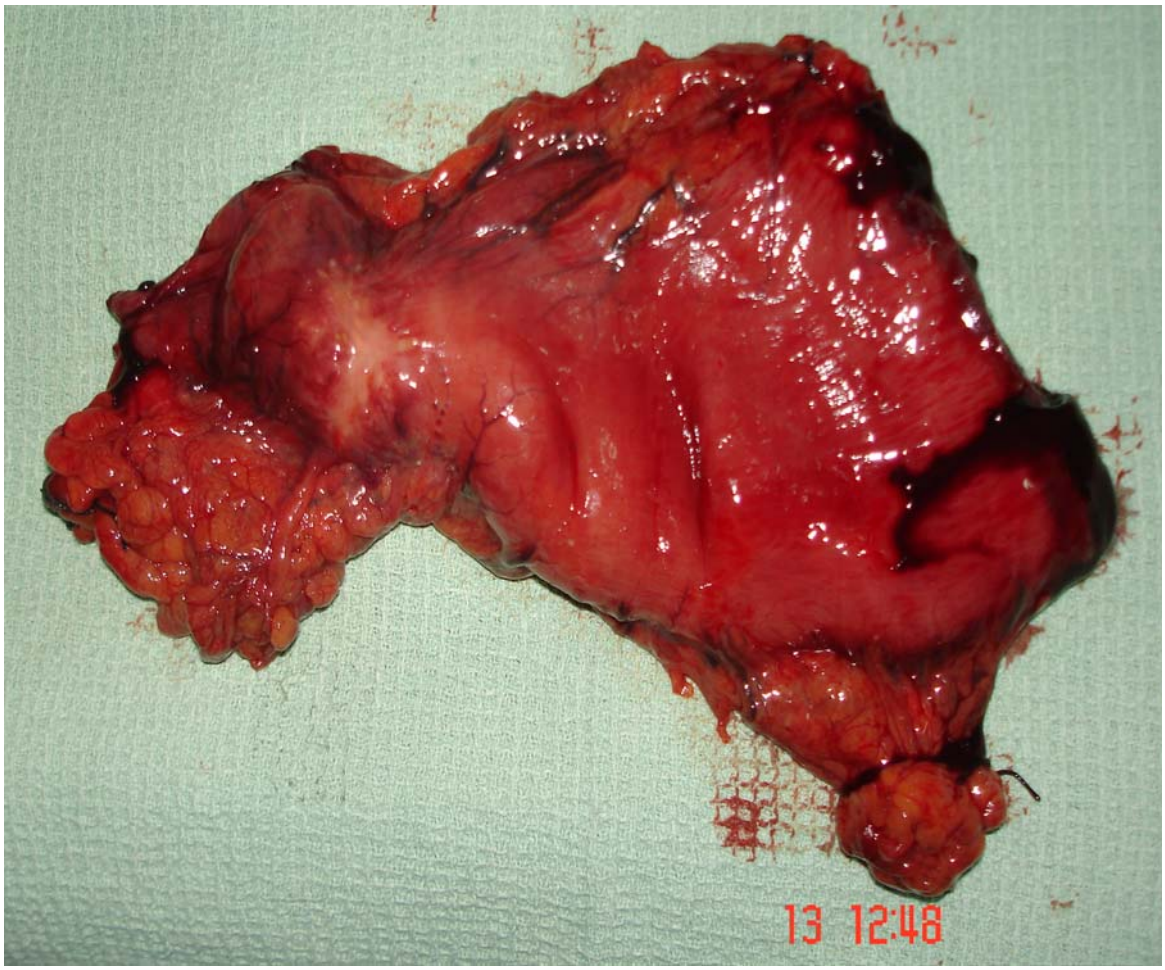



# BILLROTH II GASTROJEJUNOSTOMY





**RESECTED SPECIMEN OF CARCINOMA STOMACH**



The most important complication that follws Billroth II anastomosis is duodental blow out. This occurs mostly on fourth Postoperative day. This is due to sutures giving way at the duodenal stump. This is better prevented by draining the afferent limb with the end of the Ryle's tube. The duodenal stump is further decompressed by doing jejunojejunostomy. If there is a blowout, abdomen is reopened peritoneal cavity washed and drainage kept to create a controlled fistula. The Nutrition is maintained either parenterally or through a feeding jejunostomy. The fistula closes later.

The other complications includes Bile vomiting afferent loop syndrome, nutritional deficiencies like $B_{12}$ deficiency, iron deficiency, and dumping syndrome. For inoperable cases, palliation can also be achieved through **self expanding metallic stents.**

## MANAGEMENT OF OTHER CAUSES

i) Corrosive antral stricture

The corrosive stricture occurs most commonly in the antrum that presents as gastric outlet obstruction Retention of acid in the antrum due to pyloric sphincter spasm causes stricture. If the stricture involves only the pyloric ring, procedure of choice is pyloroplasty, usually of finney's type. If pylorus involvement is more, then ideal treatment would be an antrectomy with Billroth I Anastomosis.

1) Carcinoma of the pancreas

If the tumour in head of pancreas is resectable, Whipple procedure is done. If it is an unresectable tumour, a triple bypass procedure is completed. It comprises of cholecystojejunostomy (to relieve jaundice). Gastrojejunostomy (to relieve G.O.O) and Jejunojejunostomy lower down so that intestinal contents are diverted from biliary tree.

The management of gastric outlet obstruction due to other causes are based on established lines of management of individual cases.

# *PRESENT STUDY*

# PRESENT STUDY

## OBSERVATION

A total number of fifty (50 cases) patients were studied cases admitted in general surgical ward of KMC Hospital were taken in a random fashion of study. The exclusion criteria includes age less than 18 years and patients with a preoperative admission endoscopic diagnosis of either carcinoma of the stomach or chronic duodenal ulcer. The patients were ranging from 18 years to 80 years. The period of study was from October 2006 to October 2008. There were 35 males 15 females.

The cases were admitted on the basis of complaints comprising mainly of epigastric discomfort, vomiting and ball rolling movements.

## INVESTIGATIONS DONE

Base line investigations were done for all the patients 16 cases had clinical pallor and their hemoglobin was below 9 gm %. Complete blood chemistry including renal parameters and electrolytes were done.

LFT was done in patients with suspected malignancy.

Three patients had jaundice and had serum bilirubin greater than 2 mgm %.

No diabetic patient was found in this group.

All patients had x-ray chest PA view, abdomen and ECG. 3 cases had significant ECG changes for which cardiologist opinion was obtained.

Endoscopy was done for all patients. 12 patients had fibrotic scarred duodenum and 37 patients had a growth in stomach mainly involving the antrum. One pt had ca-head of pancreas.

Barium meal pictures were taken for cases with pyloric stenosis due to peptic ulcer disease and all 12 had dilated stomach and stasis of barium. Ultrasonogram was taken for all cases, 12 cases had liver secondaries and they were multiple. 1 case was detected to have celiac node. 2 cases were detected to have ascites in ultrasonogram.

CT scan abdomen was done for malignant cases who were affordable. Blood grouping was done for all patients.

Dehydration was corrected for all patients and 3 patients had preoperative blood transfusion. Three patients had infiltration in chest X-ray but were sputum negative for AFB or malignant cells.

All patients on the pre-operative day underwent stomach lavage through Ryle's tube. Serum electrolytes were taken on the day of surgery for intravenous management intraoperatively.

# *DISCUSSION*

# DISCUSSION

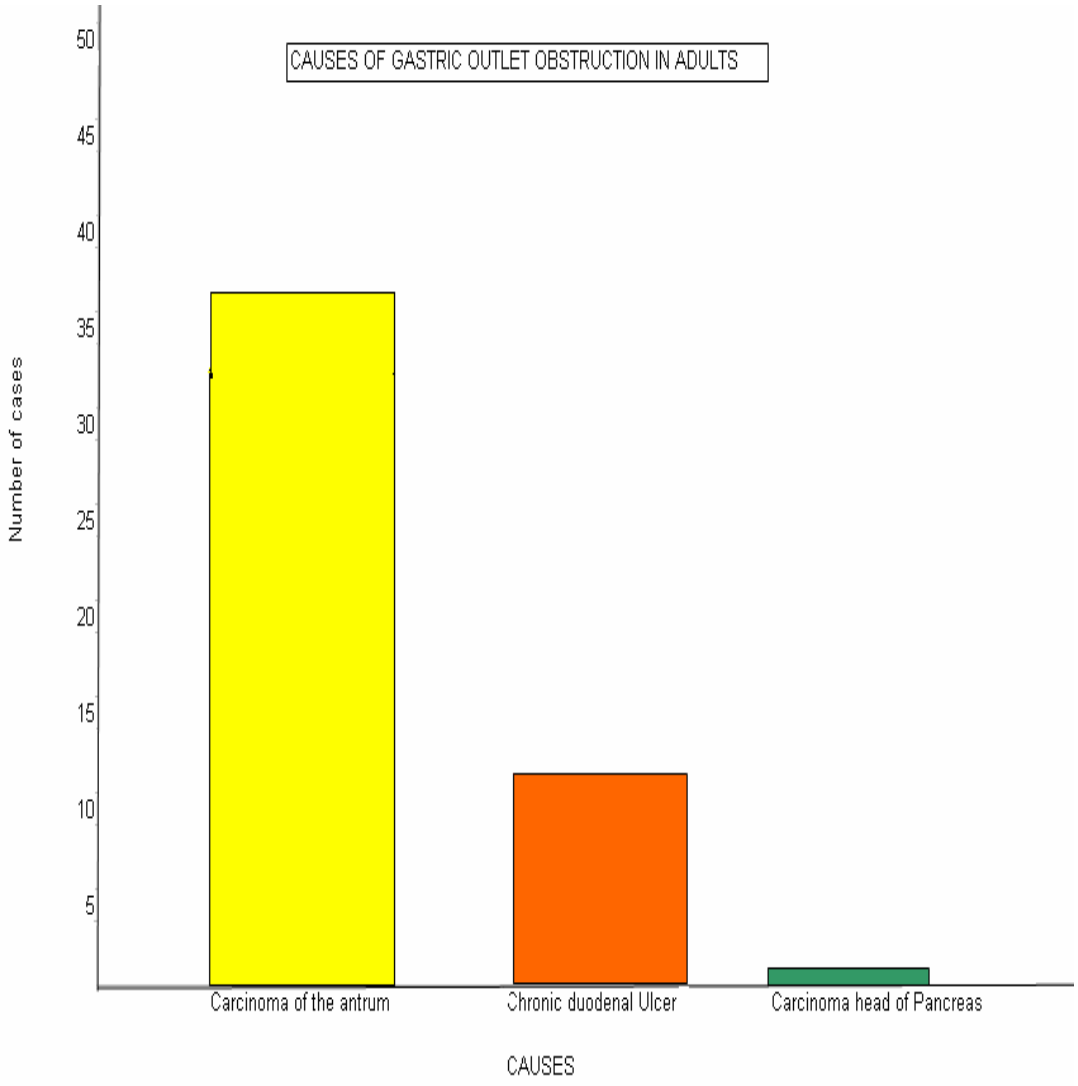
After completing all investigation of the 50 cases the cause were found to be as follows.

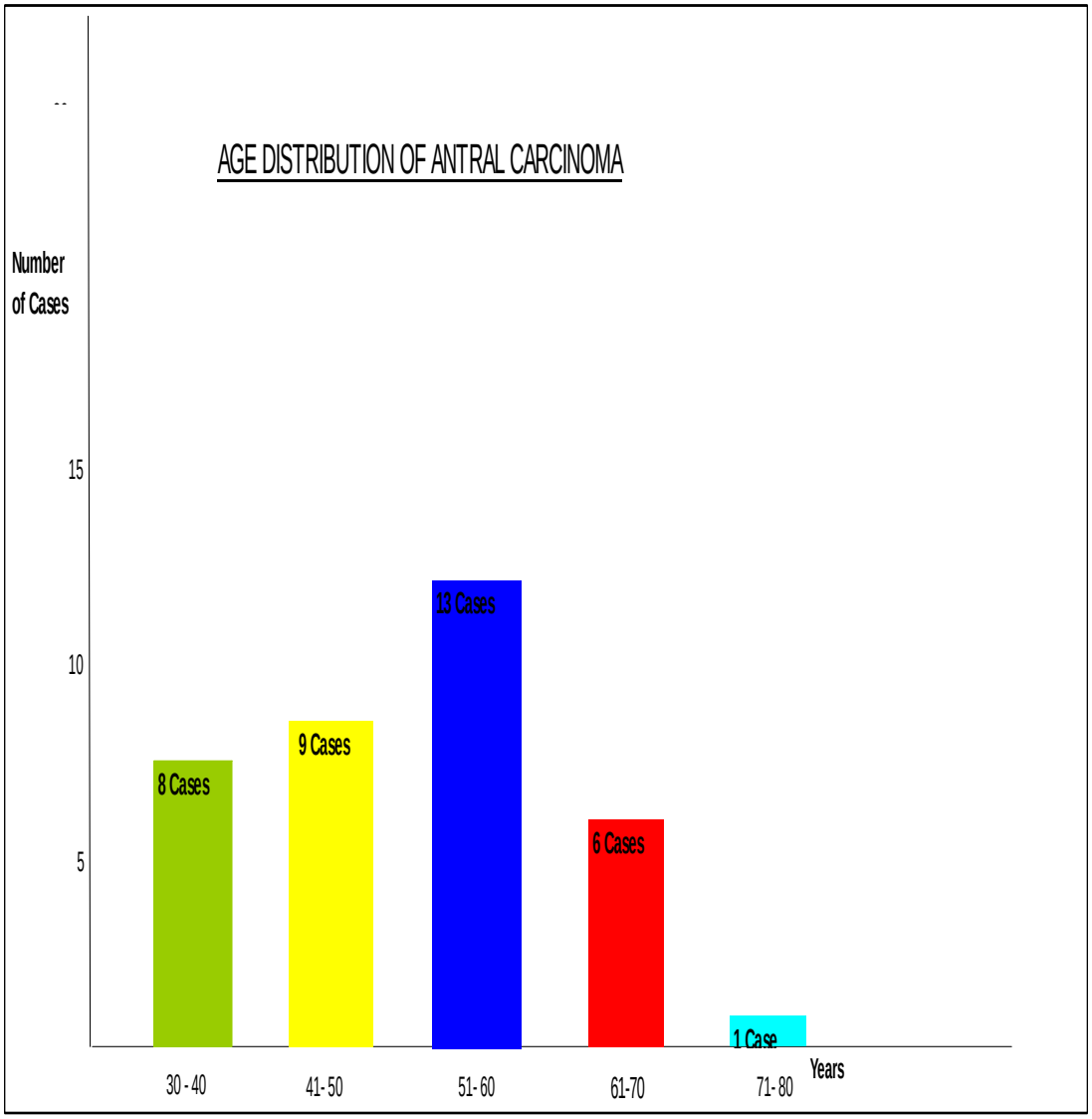
| Causes | Number of Cases |
|---|---|
| Carcinoma of the antrum | 37 |
| Chronic Duodenal Ulcer | 12 |
| Other Causes | 1 |
| **Total** | **50** |

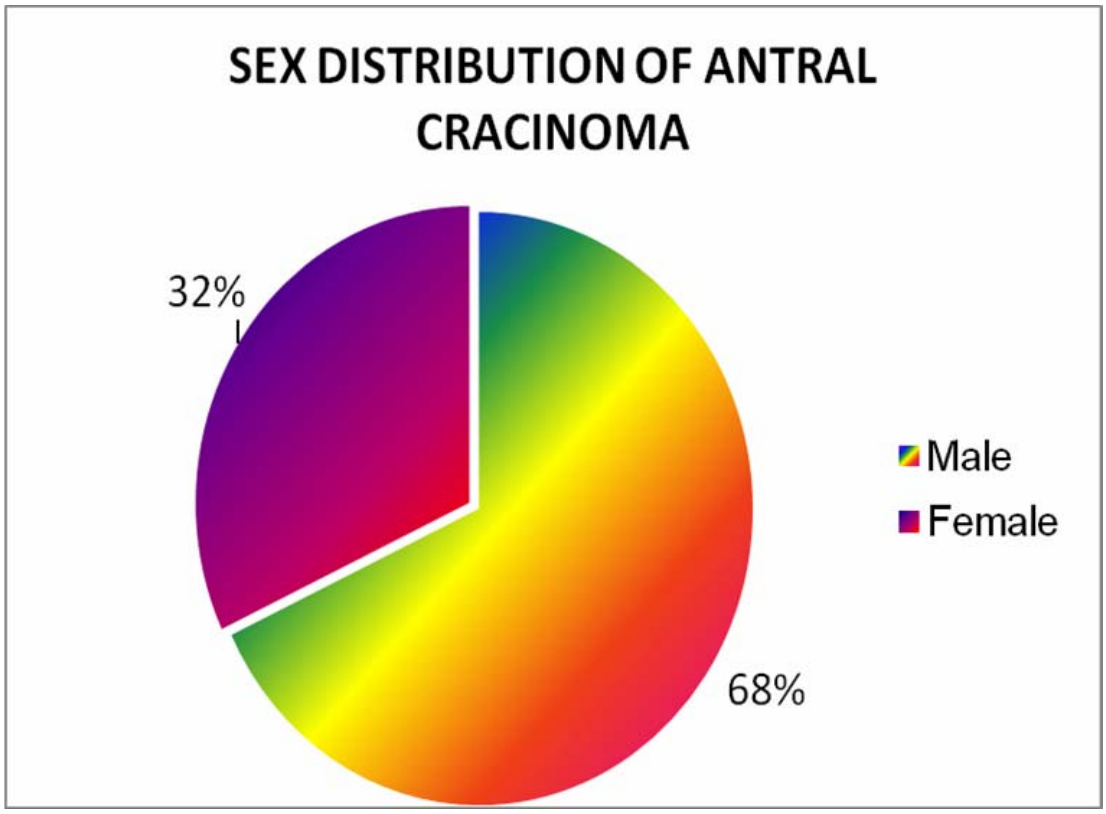
## GASTRIC OUTLET OBSTRUCTION DUE TO ANTRAL CANCER

## AGE DISTRIBUTION

| Age Interval | Number of Cases |
|---|---|
| 30-40 | 8 |
| 41-50 | 9 |
| 51-60 | 13 |
| 61-70 | 6 |
| 71-80 | 1 |
| **Total** | **37** |


CAUSES OF GASTRIC OUTLET OBSTRUCTION IN ADULTS
Number of cases
50
45
40
35
30
25
20
15
10
5
Carcinoma of the antrum
Chronic duodenal Ulcer
Carcinoma head of Pancreas
CAUSES


AGE DISTRIBUTION OF ANTRAL CARCINOMA
Number of Cases
15
10
5
8 Cases
9 Cases
13 Cases
6 Cases
1 Case
30 - 40
41- 50
51- 60
61-70
71- 80
Years


SEX DISTRIBUTION OF ANTRAL CRACINOMA
32%
68%
Male
Female

| | |
|---|---|
| **Lowest (Age of occurrence)** | **35 years** |
| **Highest (Age of occurrence)** | **80 years** |
| **Commonest Age group** | **51 to 60 years** |

**Sex Distribution CA Antrum**

| | |
|---|---|
| Male | 25 |
| Female | 12 |
| **Total** | **37** |

Sex Distribution in ANTRAL CARCINOMA

Male: 68%

Female: 32%

In this study, the incidence of carcinoma of antrum presenting with gastric outlet obstruction is high in males than females. It corresponds well with the earlier studies. The commonest age group of occurrence of malignancy was 51 – 60 years.

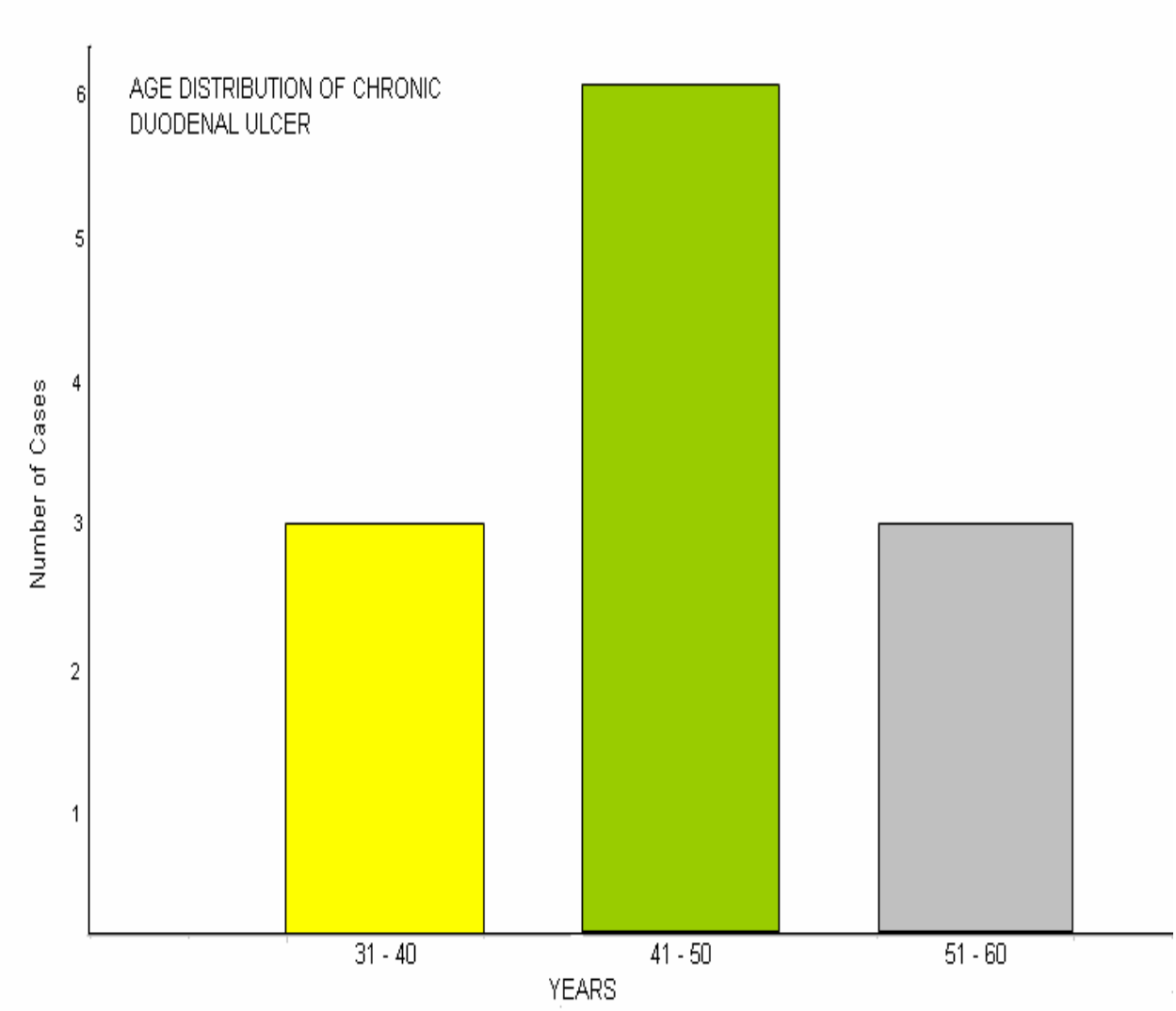
The main complaints were epigastric dyspepsia and vomiting.

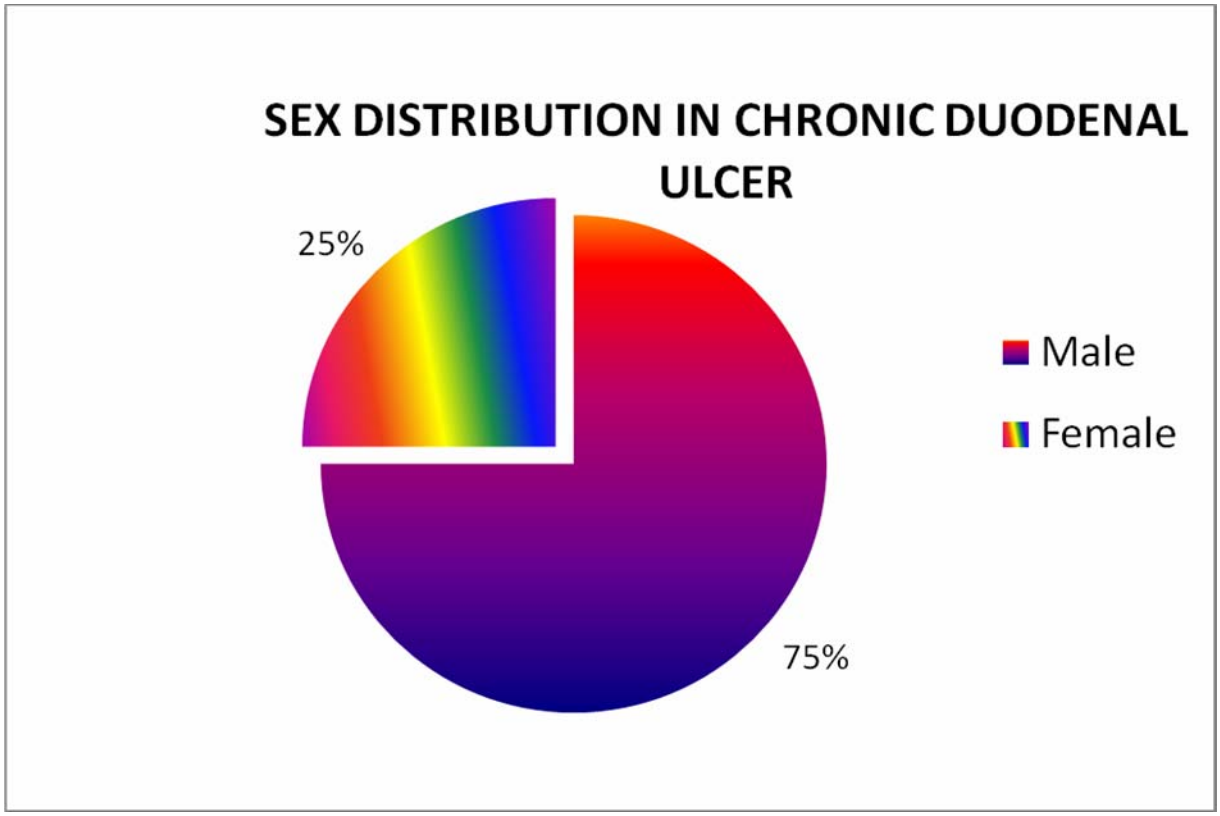
# GASTRIC OUTLET OBSTRUCTION

DUE TO CHRONIC DUODENAL ULCER

| Age Group (in years) | Number of Cases |
|---|---|
| 31– 40 | 3 |
| 41 – 50 | 6 |
| 51 - 60 | 3 |
| **Total** | **12** |

| | |
|---|---|
| Highest (Age of occurrence) | 58 |
| Lowest (Age of occurrence) | 35 |
| Commonest age of group | 41 – 50 years |


AGE DISTRIBUTION OF CHRONIC DUODENAL ULCER
Number of Cases
6
5
4
3
2
1
31 - 40
41 - 50
51 - 60
YEARS


SEX DISTRIBUTION IN CHRONIC DUODENAL ULCER
25%
75%
Male
Female

## SEX DISTRIBUTION (CHRONIC DU)

| Age Group (in years) | Number of Cases | Male | Female |
|---|---|---|---|
| 31 – 40 | 3 | 2 | 1 |
| 41 – 50 | 6 | 5 | 1 |
| 51 - 60 | 3 | 2 | 1 |
| **Total** | **12** | **9** | **3** |

Percentage:

Male : 75%

Female: 25%

The predominance of males compared well with earlier studies. It also corresponds with fact that duodenal ulcer occurs more frequently in men than women due to various factors.

Main complaints were vomiting and epigastric pain.

# TREATMENT

## PATIENTS WITH ANTRAL CANCER AND GASTRIC OUTLET OBSTRUCTION

| Sex | No. of Cases |
|---|---|
| Male | 25 |
| Female | 12 |
| **Total** | **37** |

- All patients were posted for laparotomy.
- General anesthesia was given for all patients. All cases were operated through midline incision.
- On Laparotomy

  a) Ascites : 2 patients
  b) Fixity to adjacent structures : 5 patients
  c) Liver secondaries : 12 patients

- Sub total gastrectomy with Billroth II anastomosis was done in 26 patients.
- Anterior long loop gastrojejunostomy was done in 12 patients.

## PATIENT WITH CHRONIC DU AND GASTRIC OUTLET OBSTRUCTION.

| SEX | NUMBER OF CASES |
|---|---|
| MALES | 9 |
| FEMALES | 3 |
| **TOTAL** | **12** |

- General anaesthesia was given for all patients, through midline incision abdomen was opened.
- Truncal vagotomy with short loop posterior gastrojejunostomy was done in 12 patients

## GASTRIC OUTLET OBSTRUCTION DUE TO OTHER CAUSES

There was one patient with CA head of pancreas compressing the duodenum causing GOO. Anterior long loop gastrojejunostomy was done

1) One patient had wound infection.

2) One patient had wound dehiscence

3) One patient had biliary gastritis

4) One patient had bilious vomiting.

The patients were discharged and advised to come for palliative chemotherapy.

**Comparison with recent studies**

| **Causes of Gastric outlet obstruction** | **Government Kilpauk Medical College Hospital** | **Shone Nikoomesh Smith et al** | **Mishra Dwidedi et al** | **Chowdry G.K. Dhali Banerjee et all** |
|---|---|---|---|---|
| CA Antrum | 74% | 61% | 76% | 78% |
| Chronic DU | 24% | 30% | 20% | 16% |

**Post Operative period**

In the post operative period of the patients who underwent truncal vagotomy with posterior gastrojejunostomy, two patients had wound dehiscence

The majority of the patients who underwent subtotal gastrectomy with Billroth type II anastomosis and anterior gastrojejunostomy had uneventful postoperative period except 4 patients.

5) One patient had wound infection.
6) One patient had wound dehiscence
7) One patient had biliary gastritis
8) One patient had bilious vomiting.

The patients were discharged and advised to come for palliative chemotherapy.

# *CONCLUSION*

# CONCLUSION

From this study of 50 adult cases 34 cases were due to malignancy of the antrum of stomach i.e., 74% and 12 cases were due to chronic duodenal ulcer i.e., 24%. So with the advent of recent anti ulcer drugs the commonest cause of gastric outlet obstruction is cancer antrum of stomach, which has compared well with the recent studies.

In carcinoma of stomach with gastric outlet obstruction males are 26 in number i.e., 68% and in chronic duodenal ulcer with gastric outlet obstruction males are 12 in number i.e., 75%

Commonest age group for cancer antrum with gastric outlet obstruction is 51-60 years and that of Chronic DU with gastric outlet obstruction is 31-41 years.

Oesophagogastro duodenoscopy (OGD) is a valuable tool in diagnosing all the causes.

Although proximal gastric carcinoma in on rise carcinoma stomach due to pyloric antral growth remains a leading cause of gastric outlet obstruction.

**In our hospital the first two common causes of gastric oulet obstruction and surgery as follows**

| | | |
|---|---|---|
| For carcinoma of antrum | - | Subtotal gastrectomy + Billroth II gastojejunostomy |
| For chronic duodenal ulcer | - | Truncal vagotomy +Billroth II gastrojejunostomy |

# *PROFORMA*

## PROFORMA

Name :

Age :

Sex :

IP No.:

## PRESENTING HISTORY

**a) Vomiting**

- Colour and nature of the vomitus/ projectile type
- At late part of day/ nocturnal
- Stasis of food which was taken 12-24 hours before

**b) Epigastric Pain**

- Character
- Relieving with vomiting

**c) H/o Haemetemesis / malena**

**d) H/o loss of weight / appetite**

**PAST HISTORY**

- H/o of old ulcer disease

**O/E**

- Incterus
- Pallor
- Pedal edema
- Pulse rate
- Blood pressure
- Signs of dehydration

**P/A INSPECTION**

- VGP
- Mass

Palpation : Mass

Organomegaly / Hapatometgaly

Percussion : shifting dullness

Fluid thrill

Auscultopercussion for dilated stomach

Succession splash

other system Examination

cardiovascular system

respiratory system

central nervous system

**INVESTIGATIONS**

Blood complete haemogram

renal parameters

chest X-ray PA view

abdominal X-ray AP view

electrocardiogram

ultrasonography of the abdomen

oesophagogastroduodenoscopy with biopsy

CT abdomen contrast study

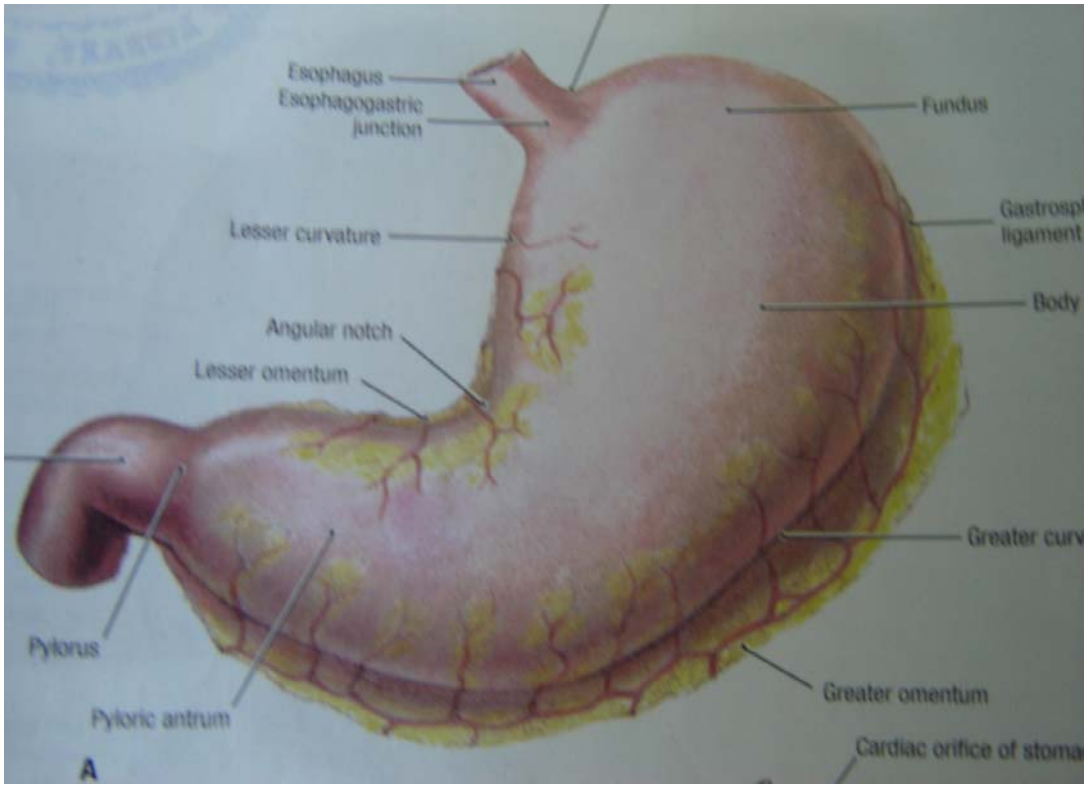
Bone scan

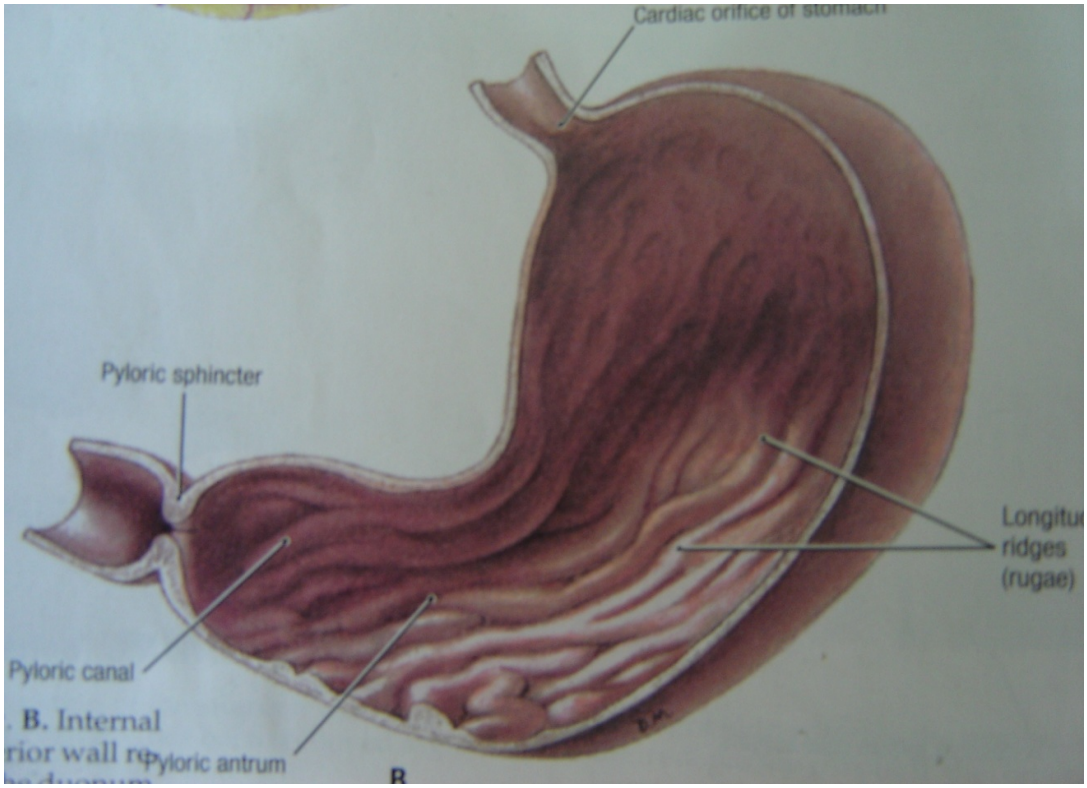
**SURGERY**

1. Intra operative findings
2. Procedure

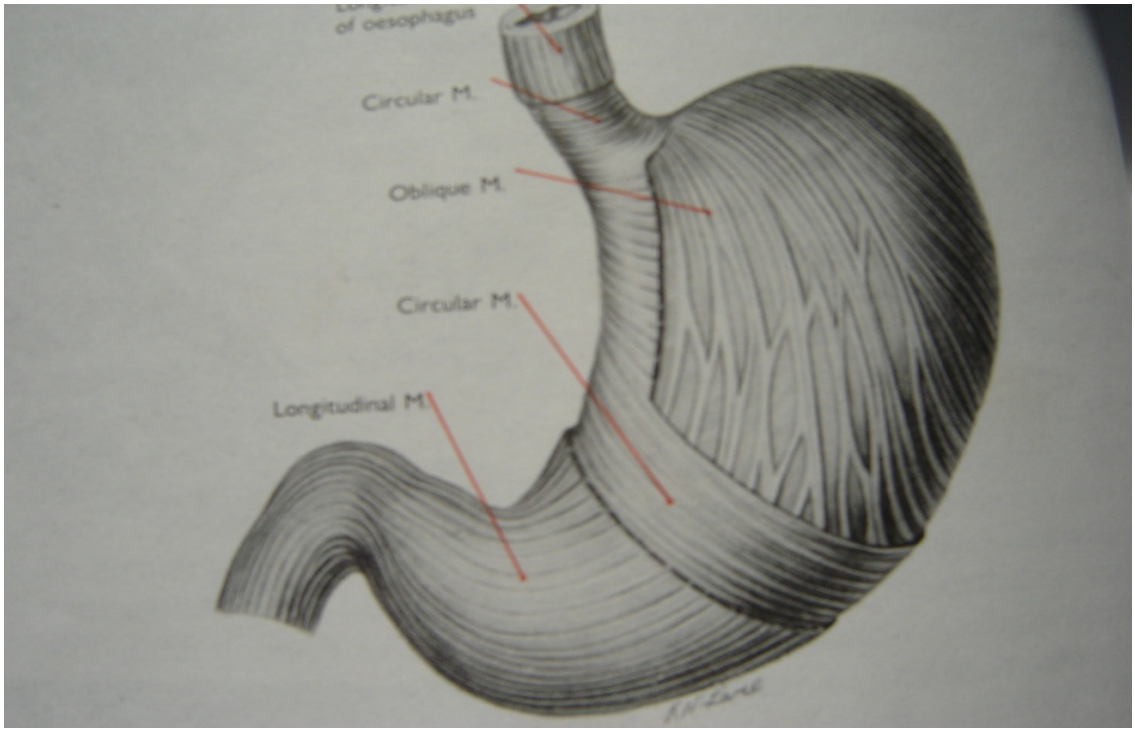
**COMPLICATIONS (IF ANY)**

# ANATOMY OF STOMACH

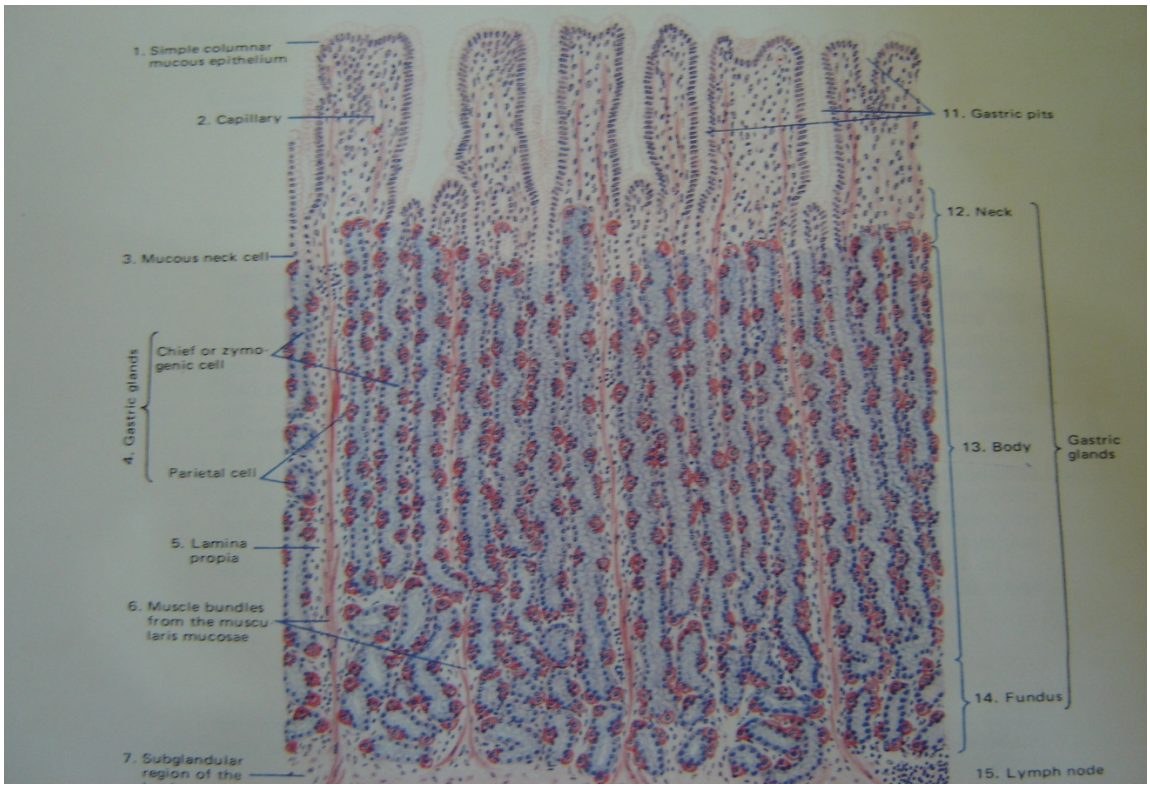


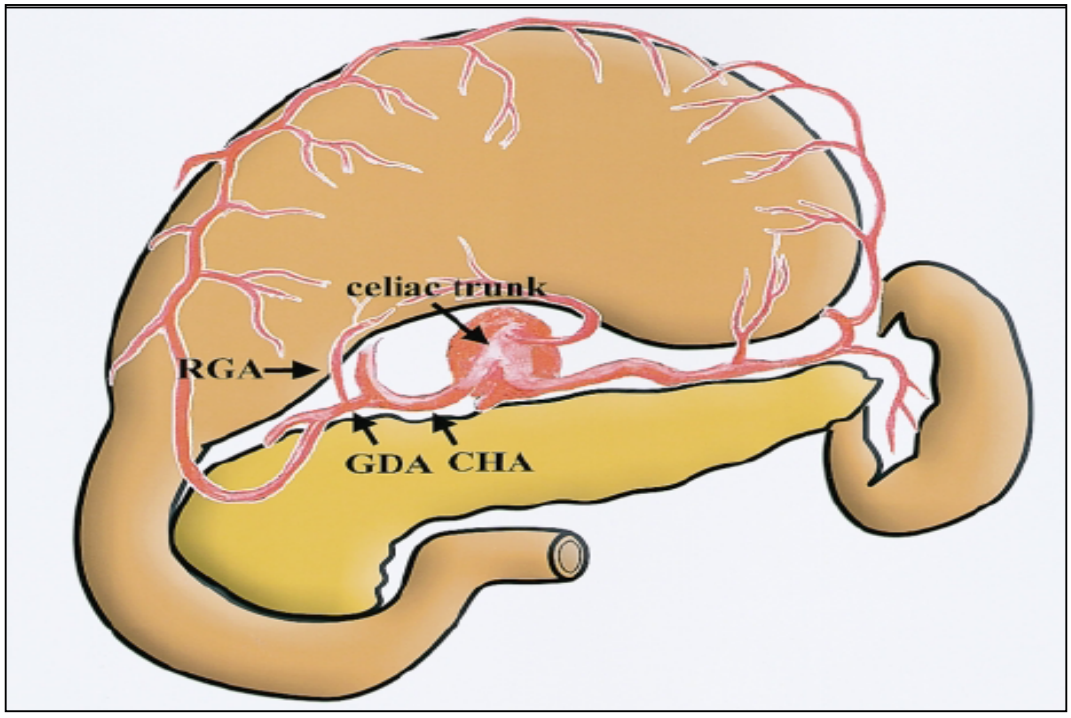
# ANTERIOR VIEW FO THE STOMACH

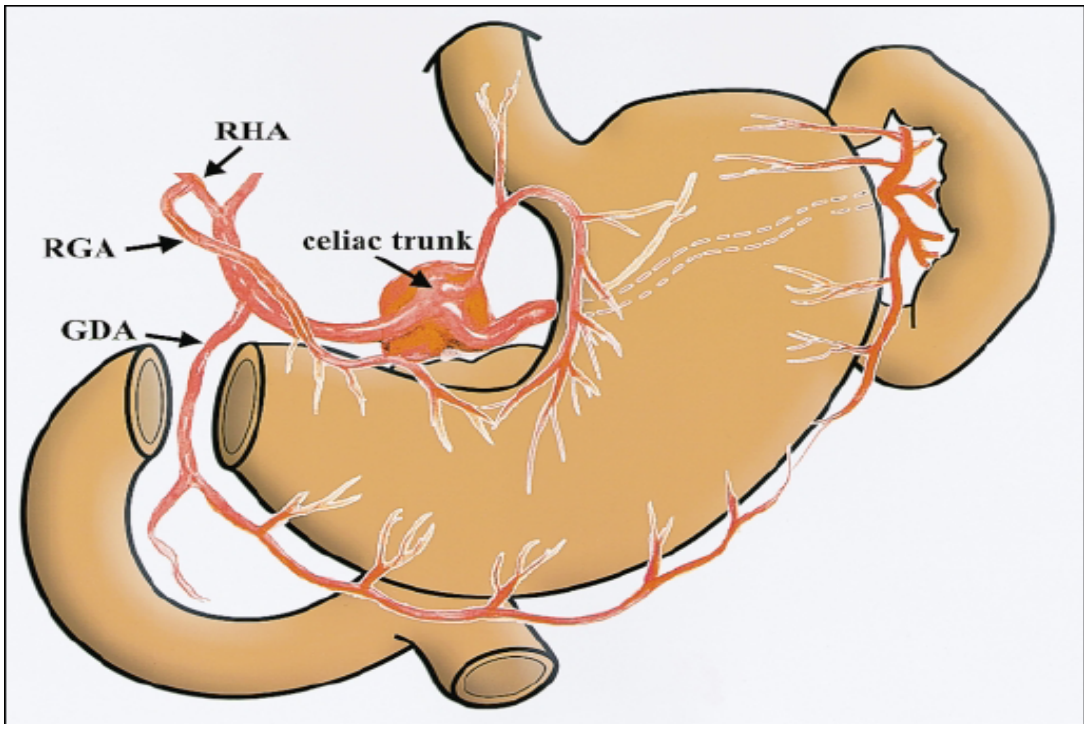



# NORMAL HISTOLOGY OF THE STOMACH

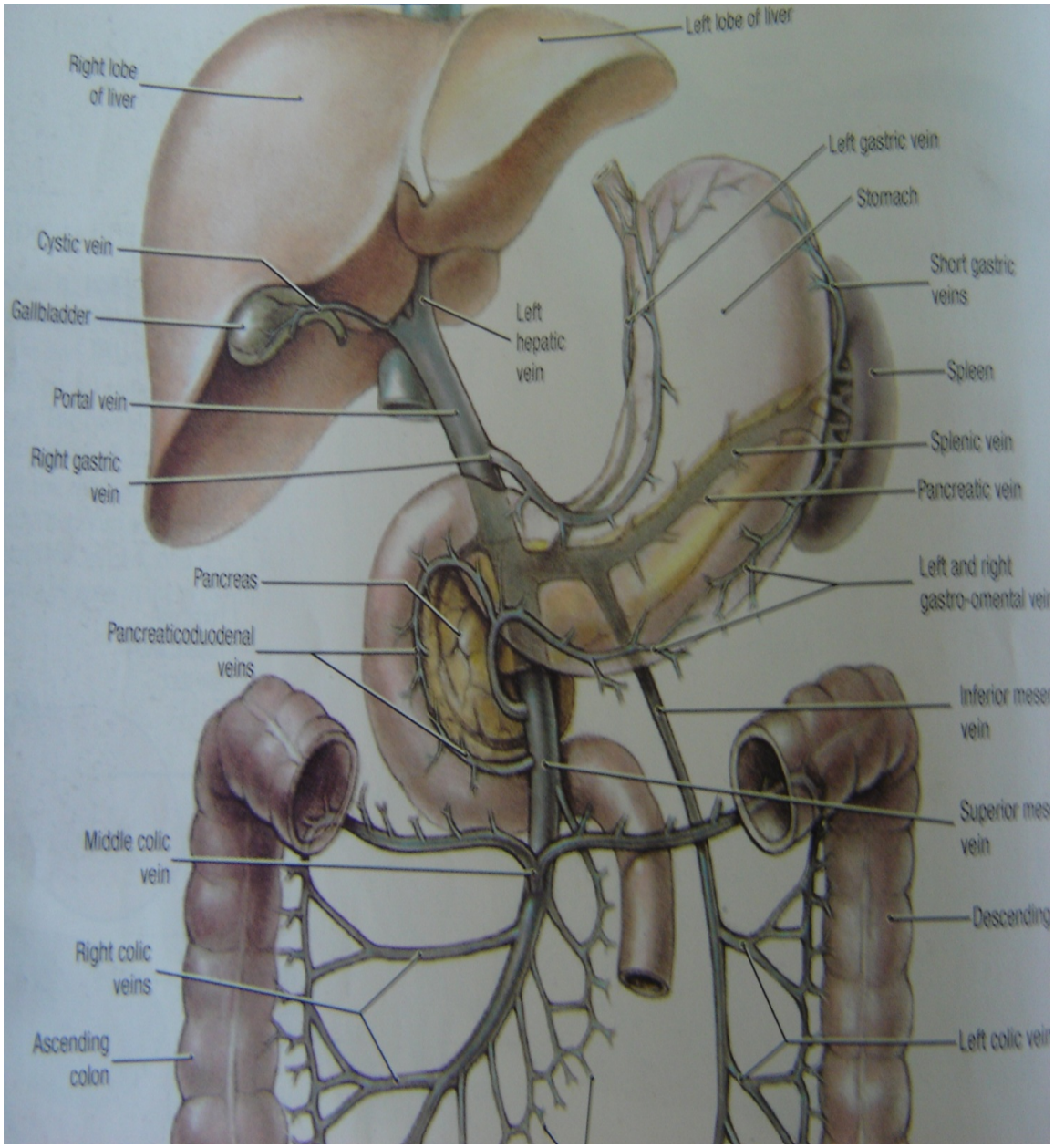


# VASCULAR SUPPLY OF THE STOMACH

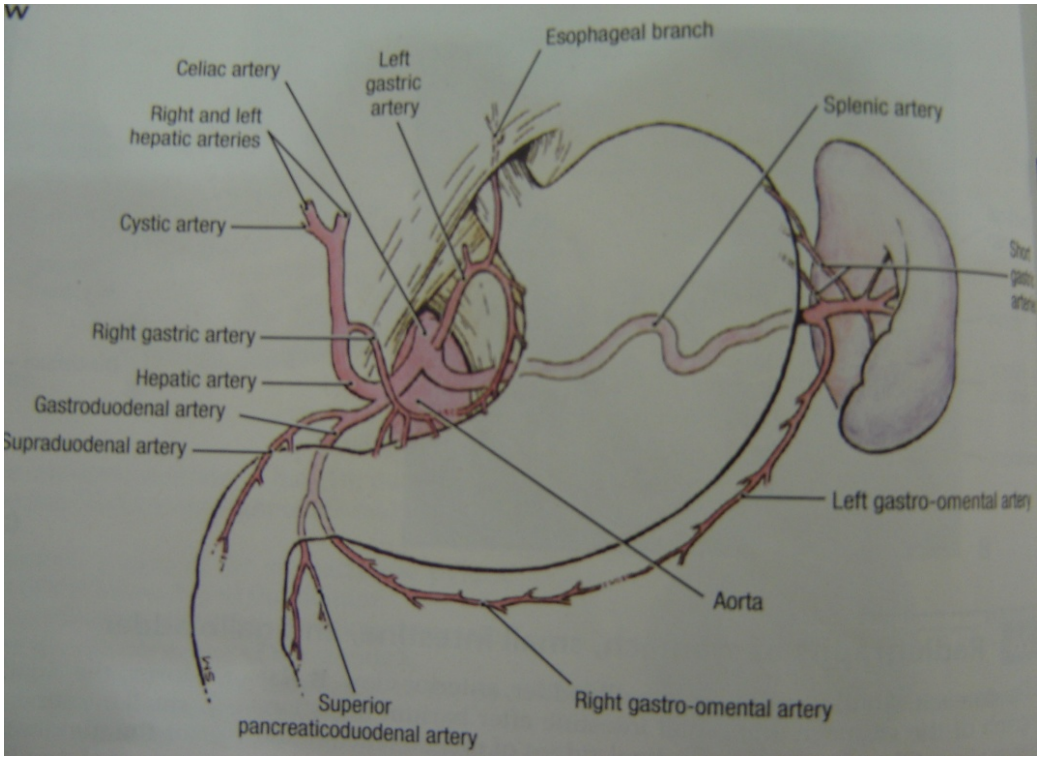


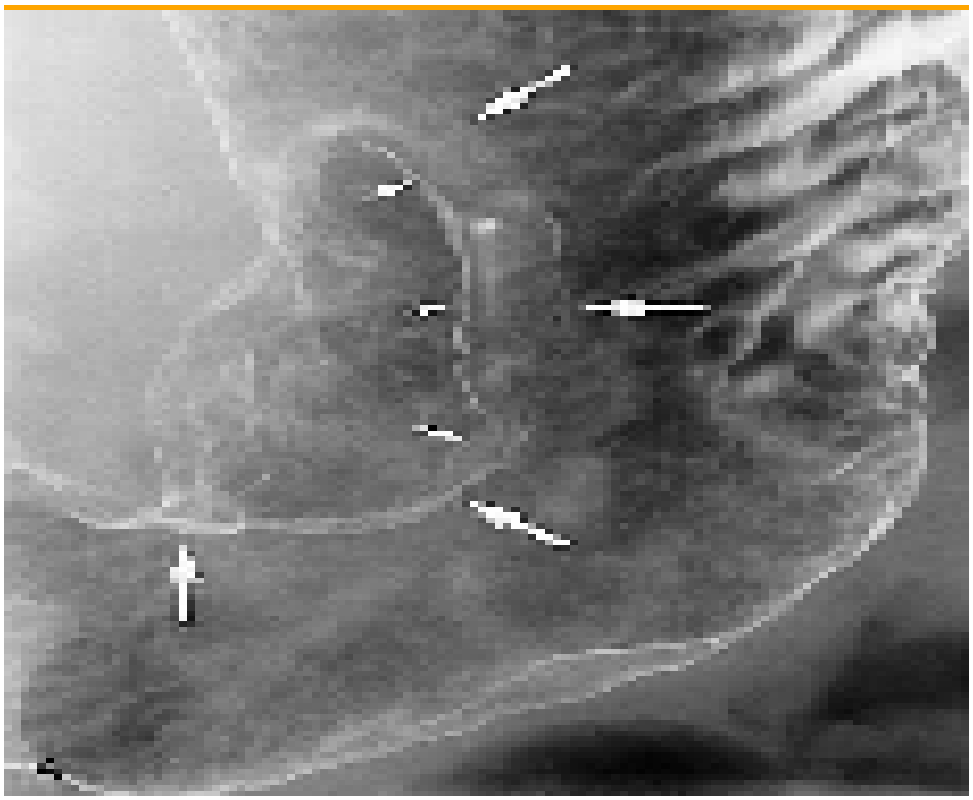
# ANTERIOR RELATIONS OF THE STOMACH



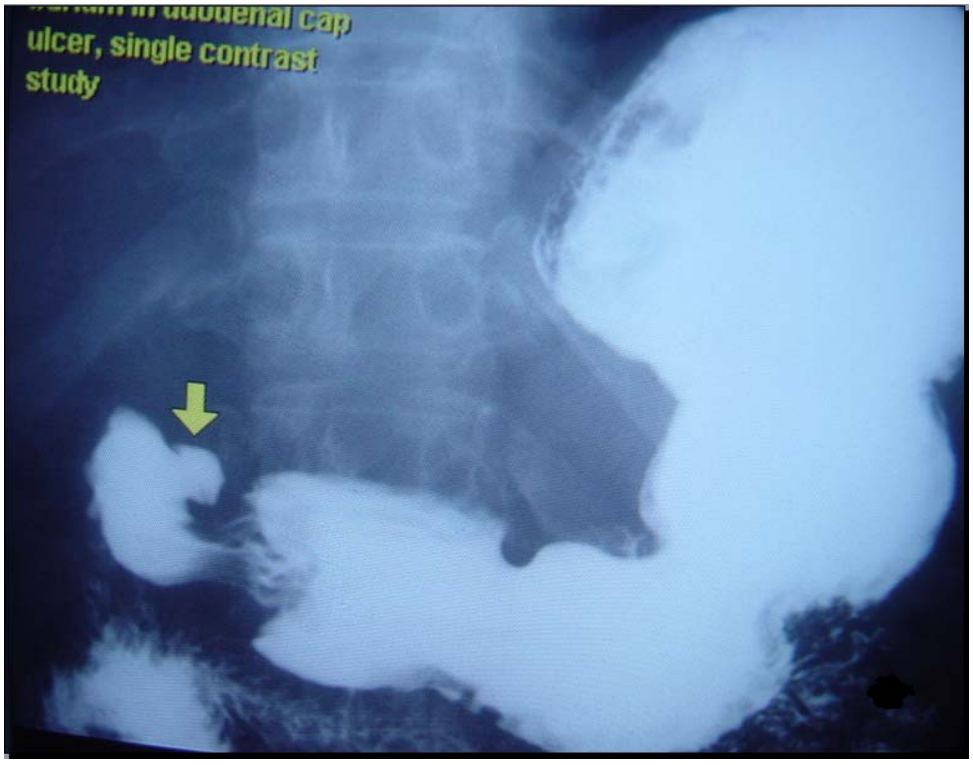
# VASCULAR AND LYMPHATIC SUPPLY OF THE STOMACH



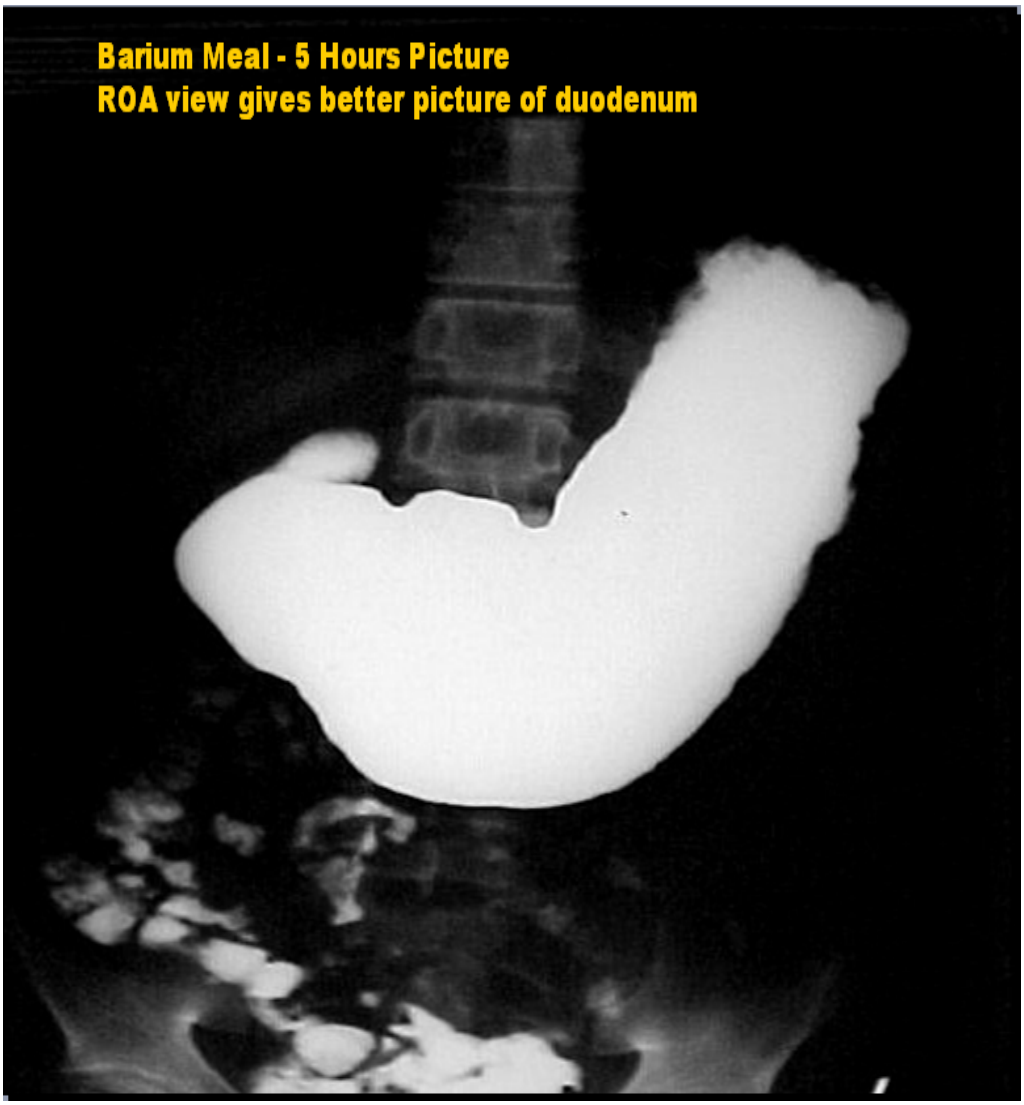

**BARIUM STUDY- SHOWING GASTRIC OUTLET OBSTRUCTION**



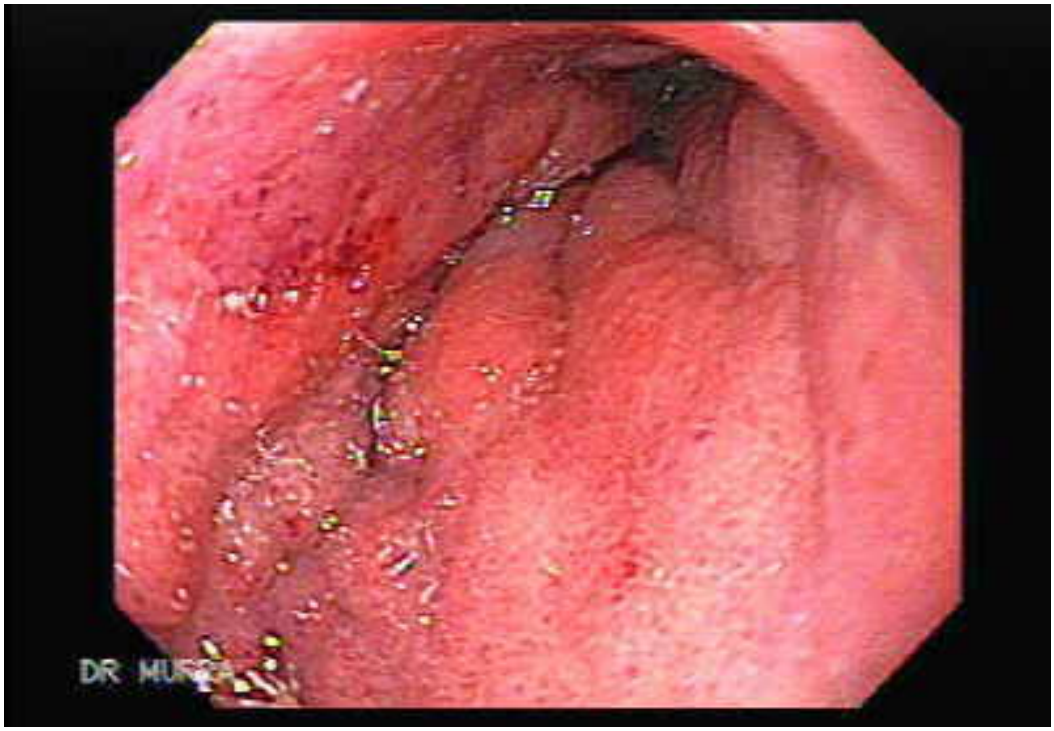
**BARIUM STUDY DEFORMED DUODENAL BULB**



**BARIUM FILM SHOWING GASTRIC OUTLET OBSTRUCTION**

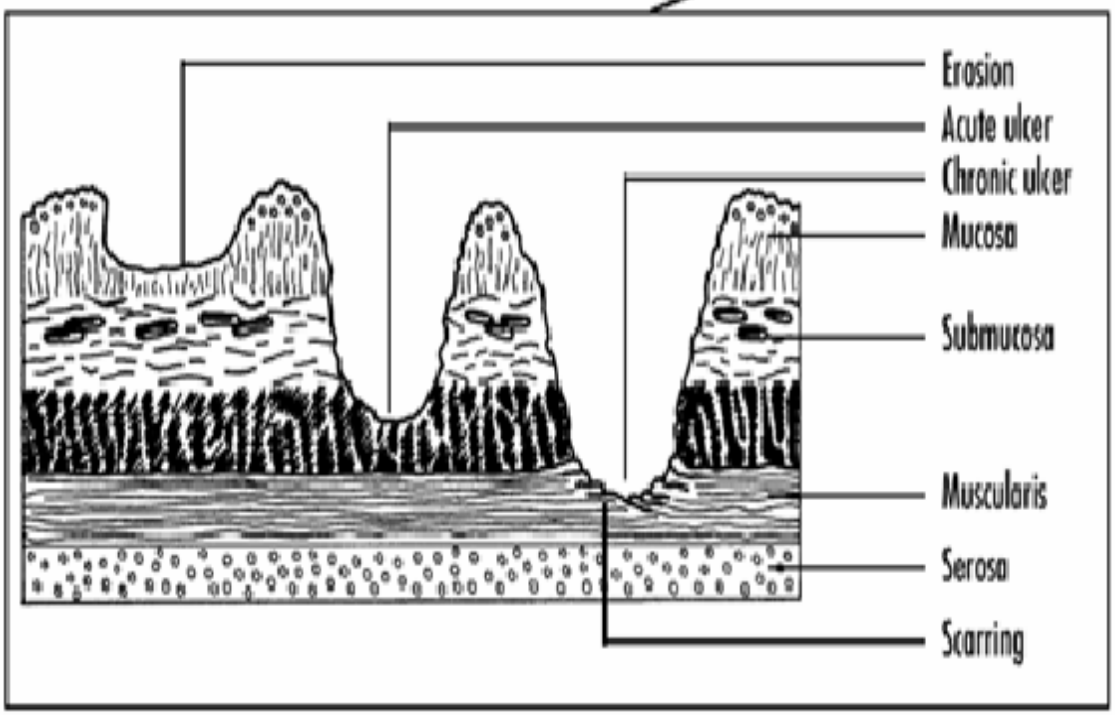


**ENDOSCOPY SHOWING TUMOUR CAUSING GASTRIC OUTLET OBSTRUCTION**

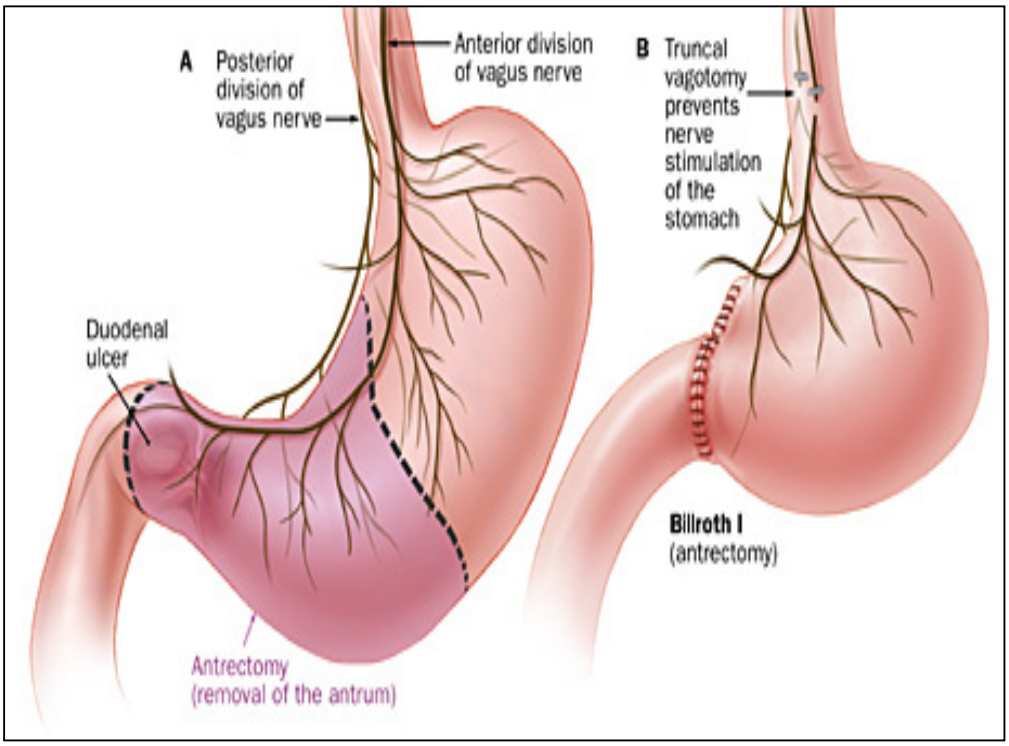


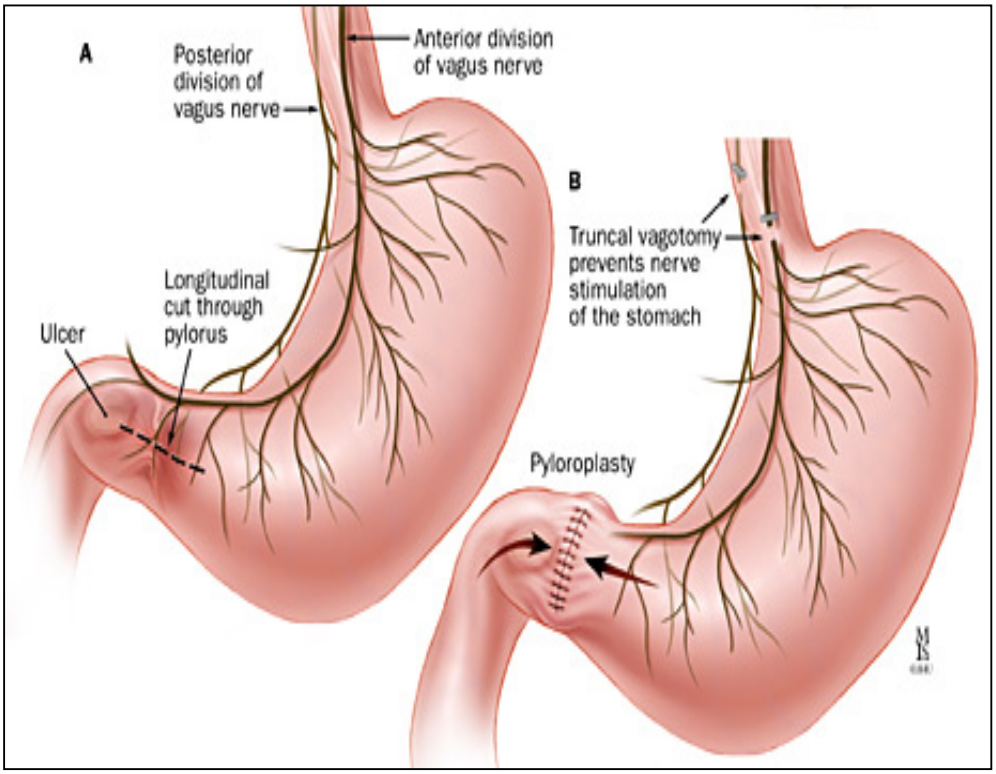
**COMPLETE GASTRIC OUTLET OBSTRUCTION BY THE TUMOUR**

A gastrointestinal lesion is not necessarily an ulcer. Lesions that don't extend below the mucosal lining (epithelium) are called erosions. Lesions of both acute and chronic ulcers can extend through the epithelium and perforate the stomach wall. Chronic ulcers also have scar tissue at the base.

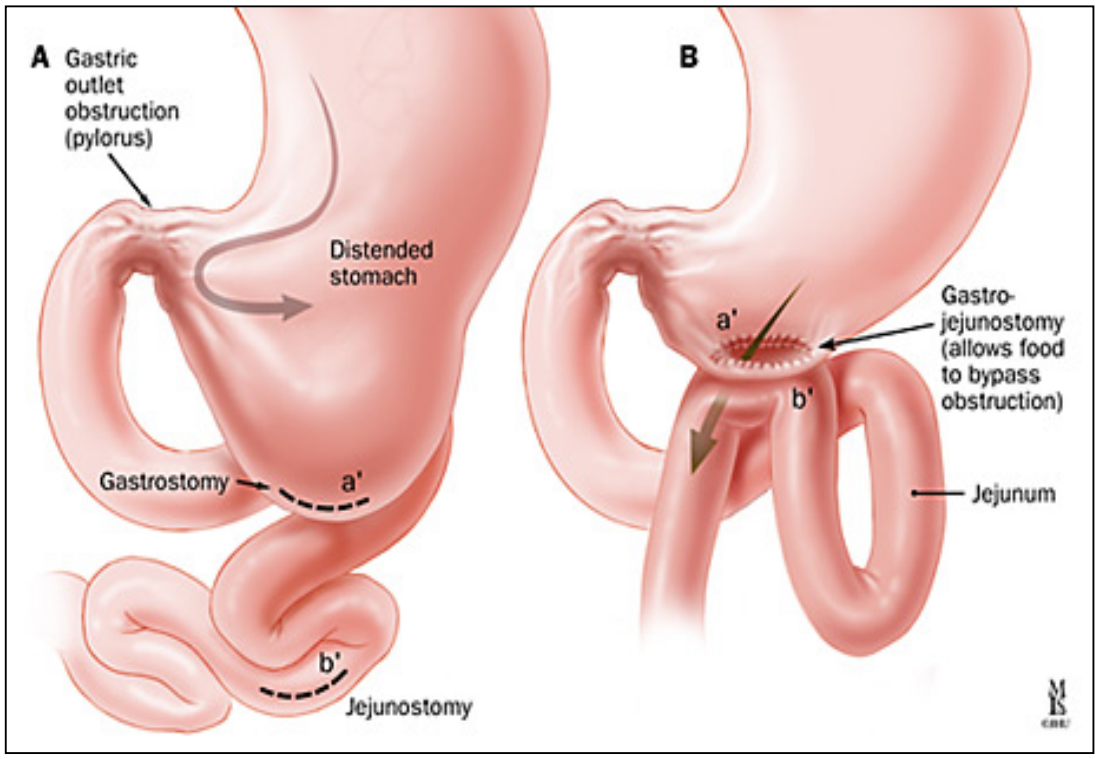


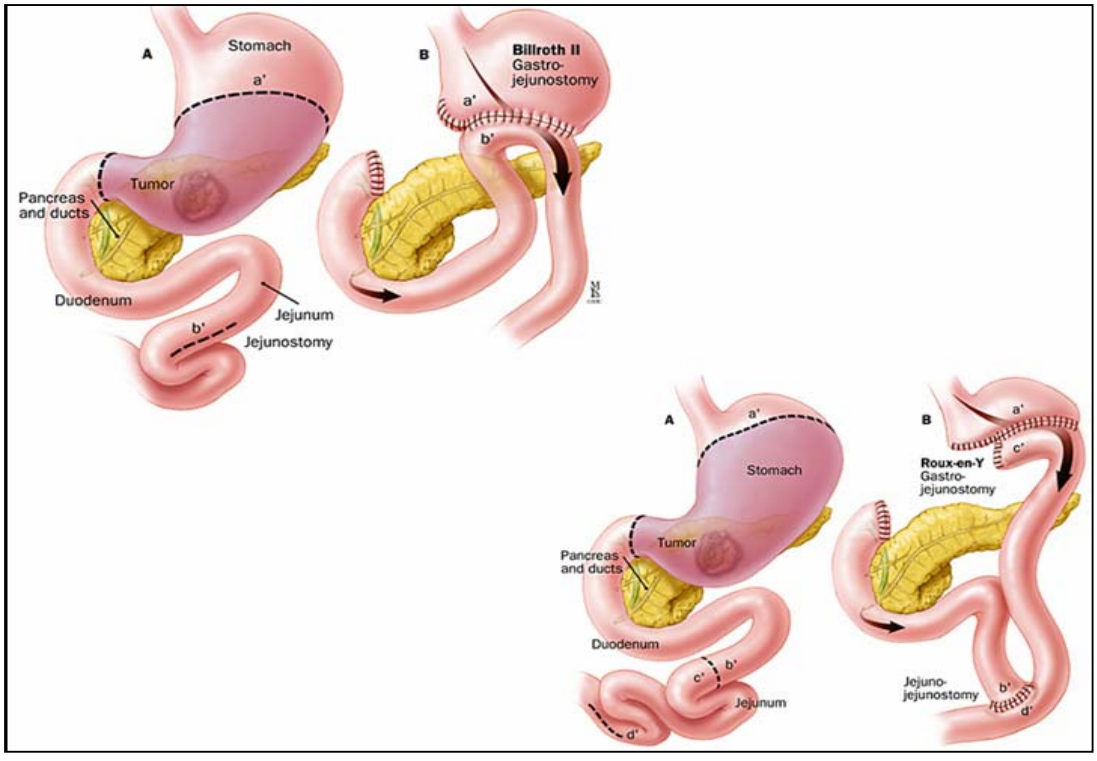
# BILLROTH I GASTRECTOMY

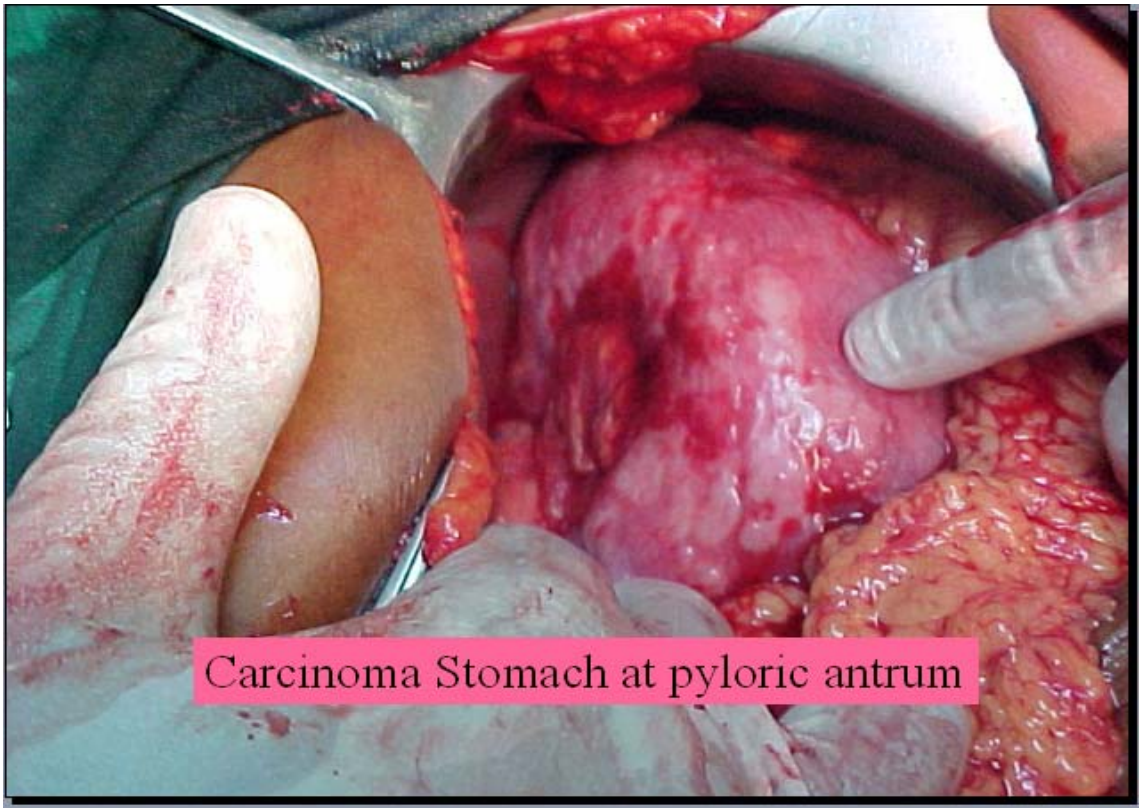


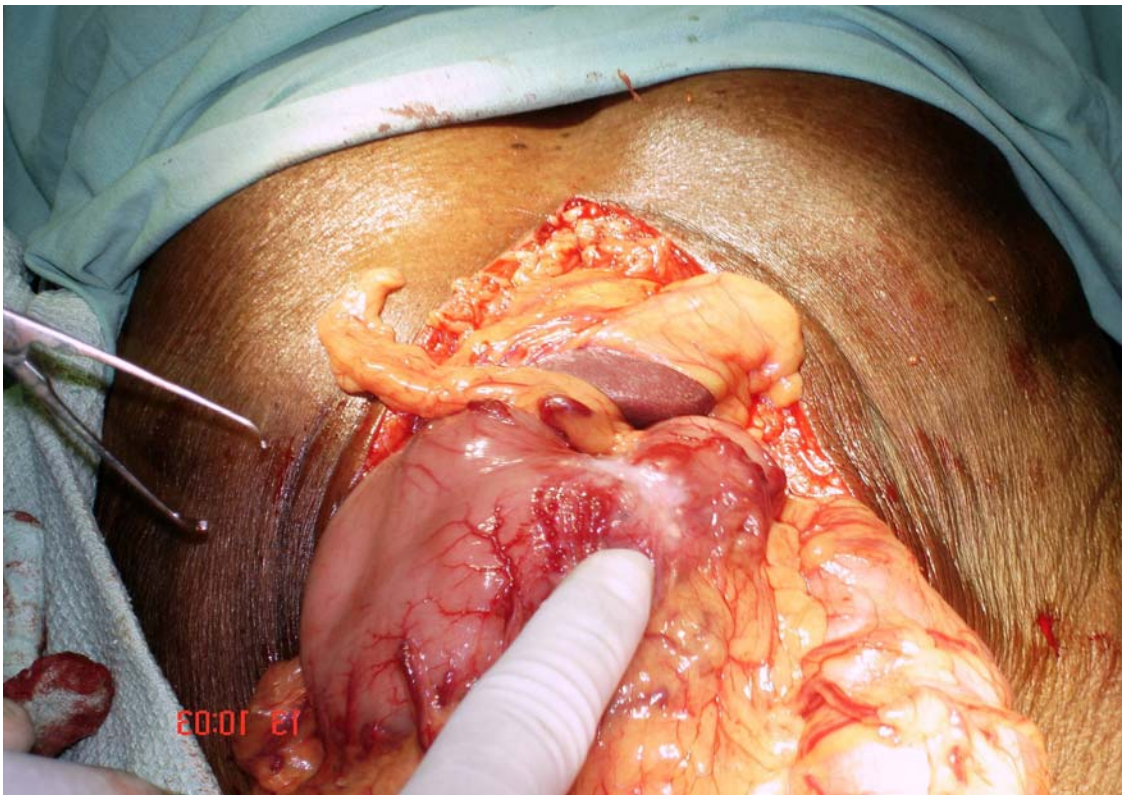
# BILLROTH II GASTRECTOMY



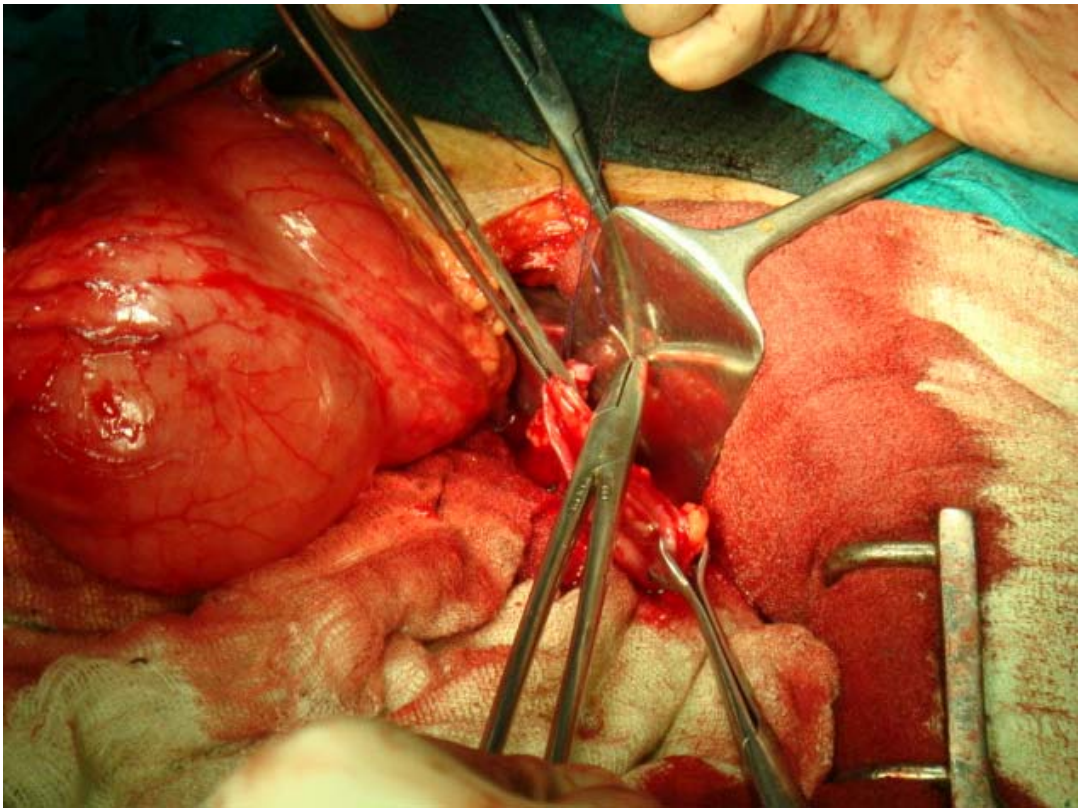



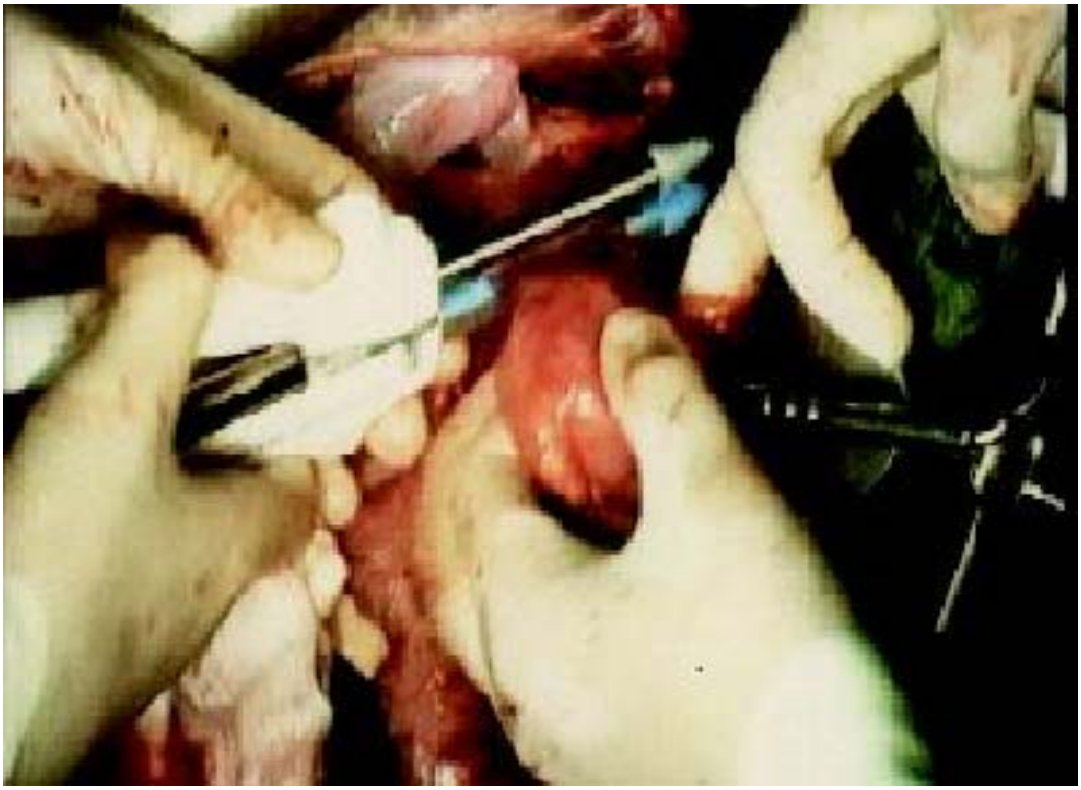



**CARCINOMA STOMACH AT PYLORIC ANTRUM**

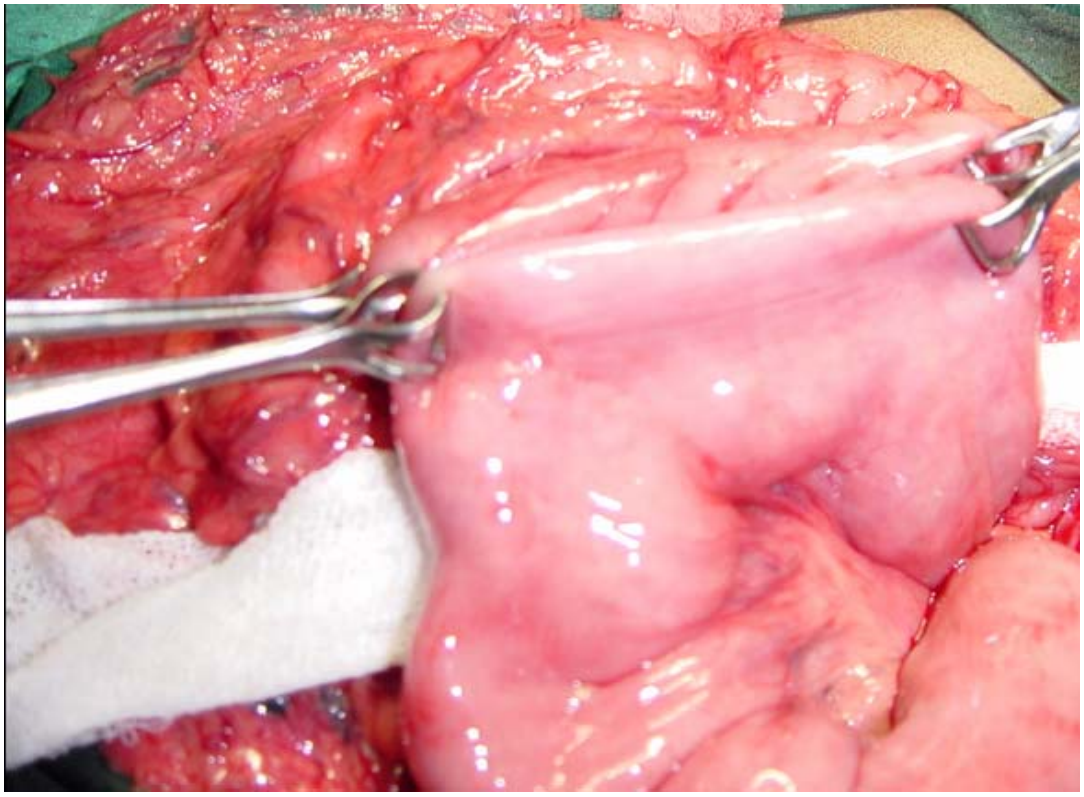


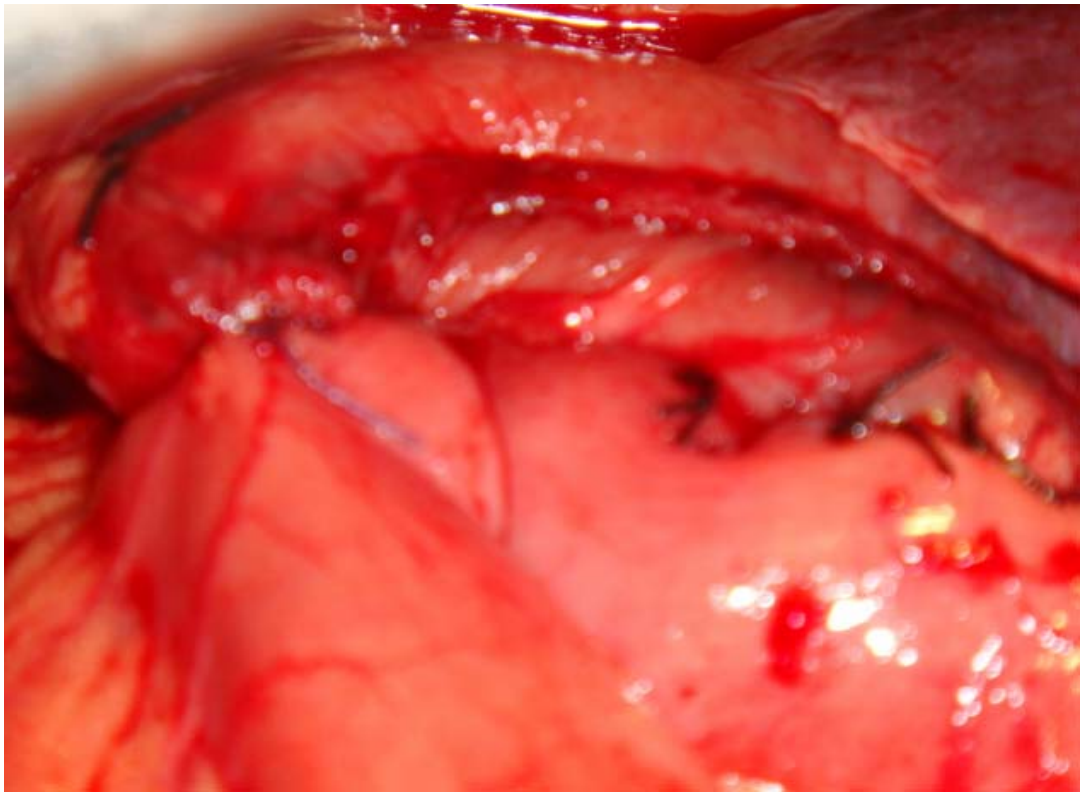
# DUODENAL STUMP CLOSURE

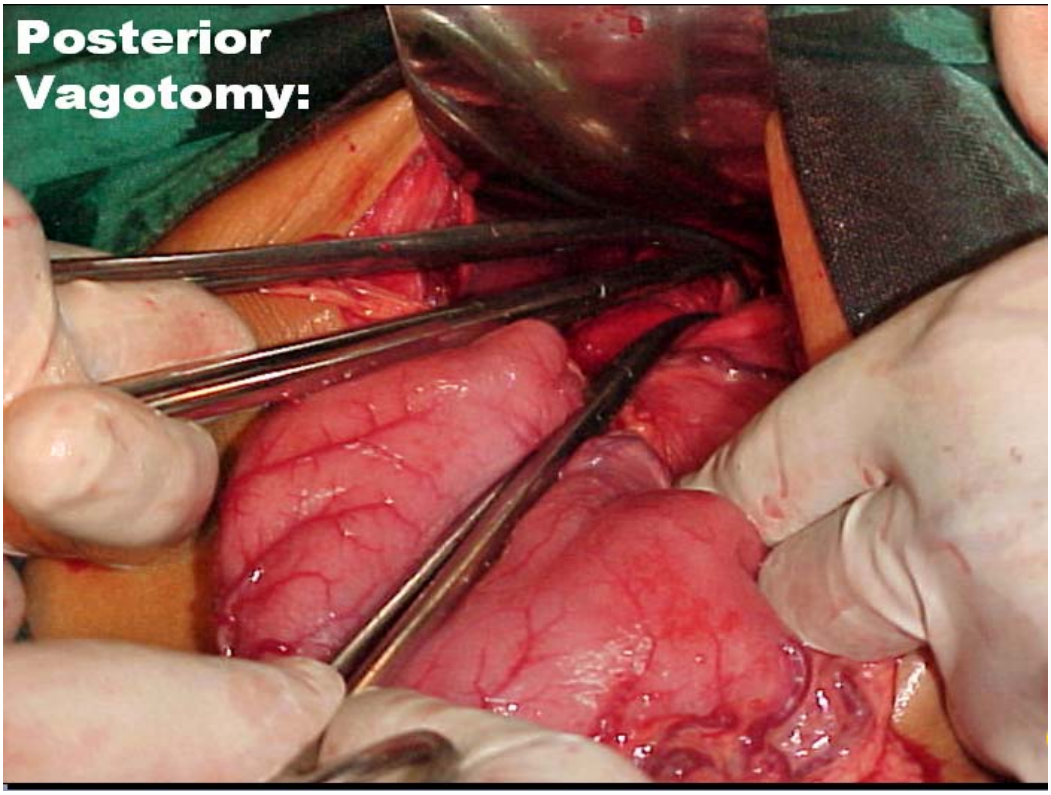




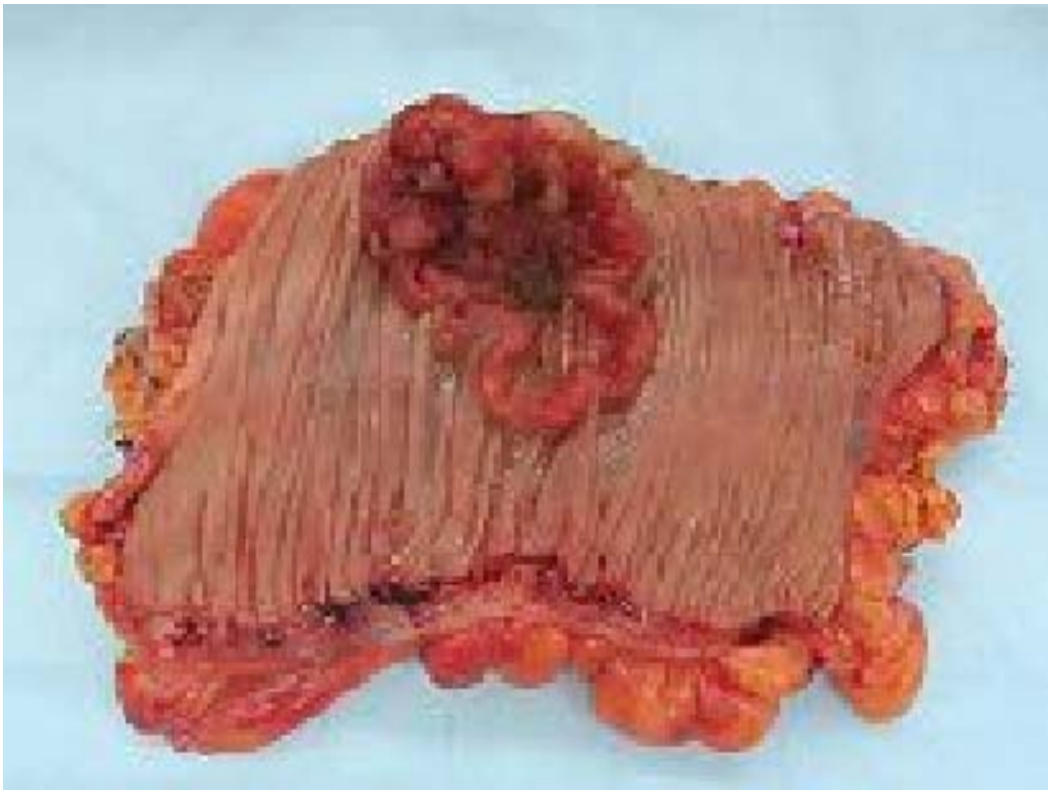
# BILLROTH II GASTROJEJUNOSTOMY



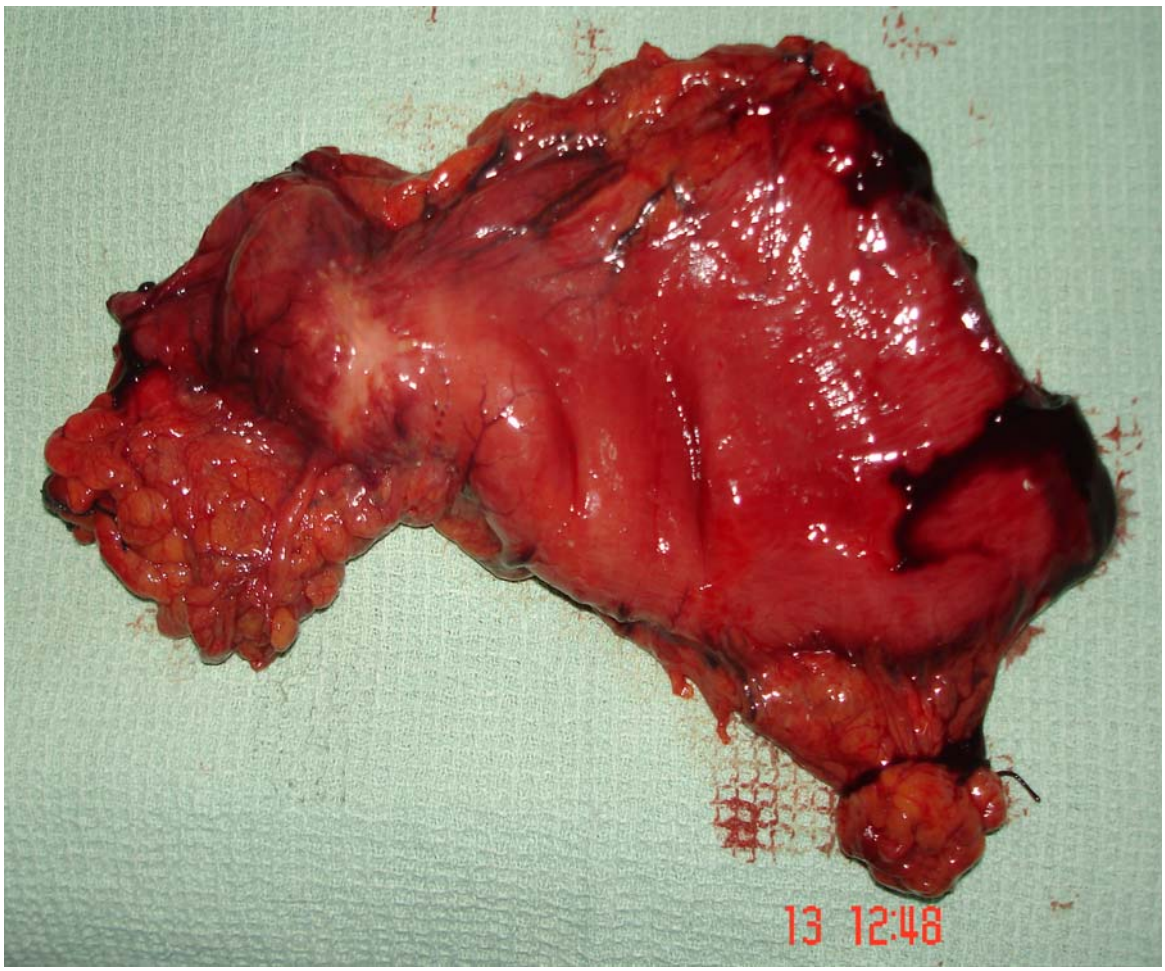


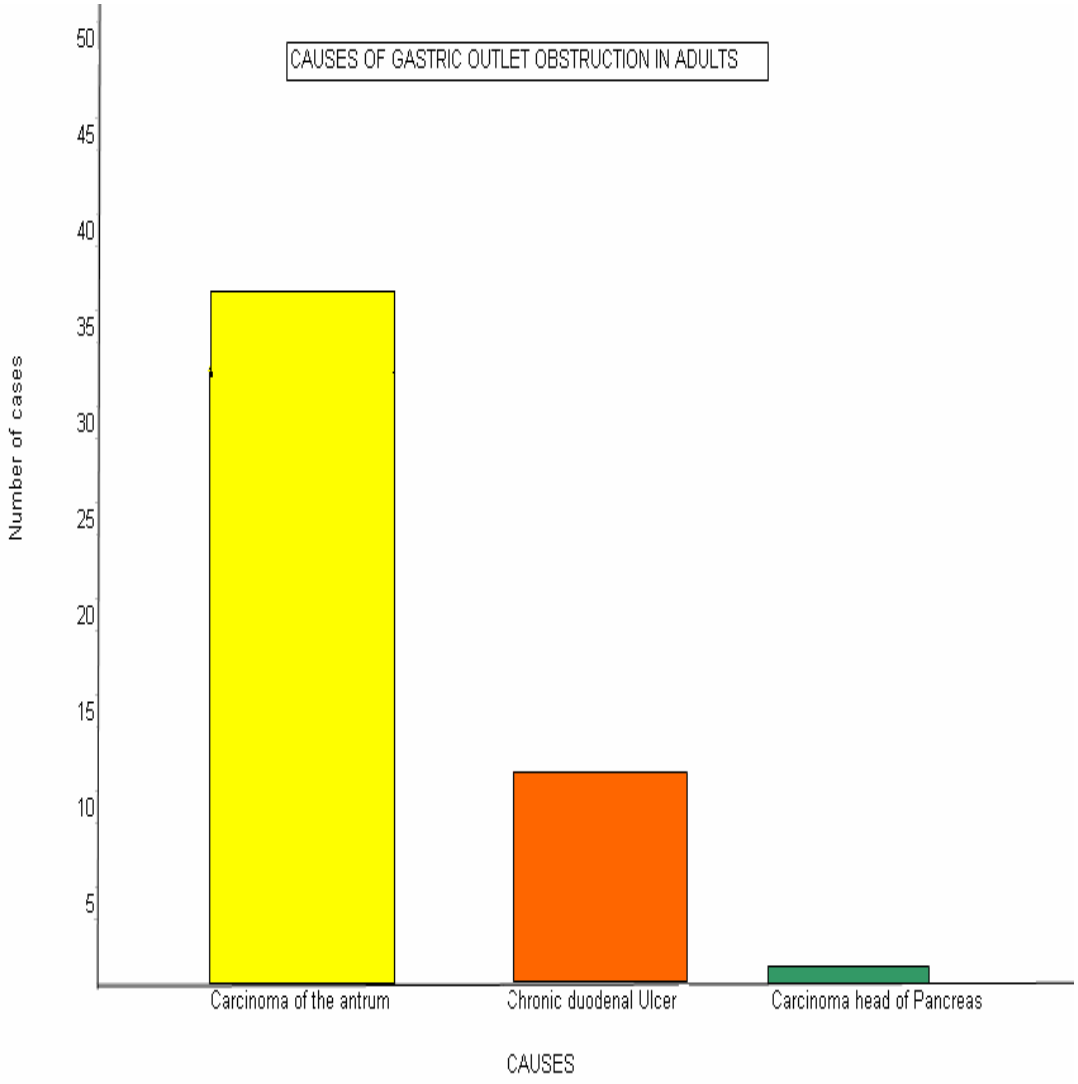


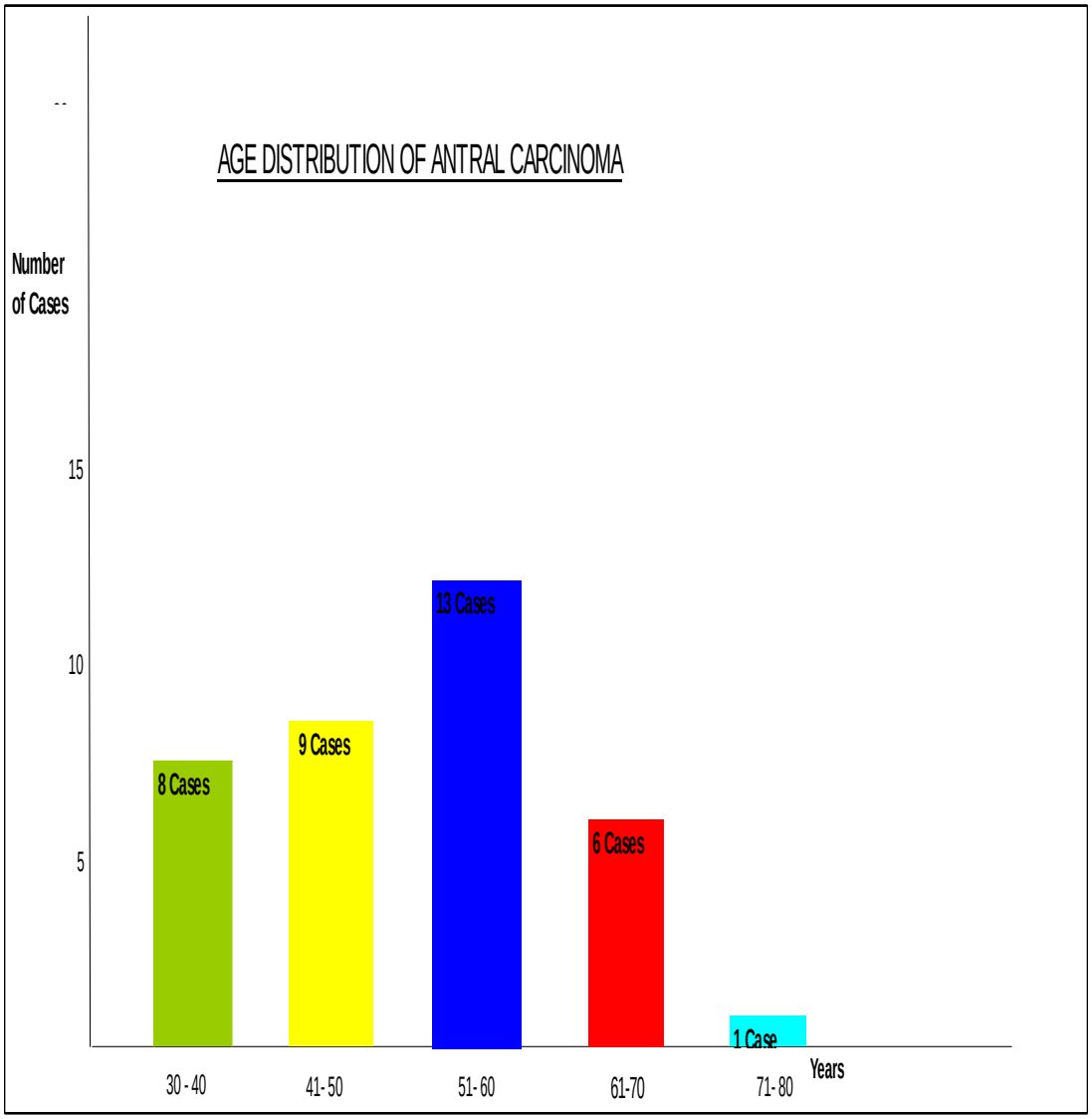

**RESECTED SPECIMEN OF CARCINOMA STOMACH**

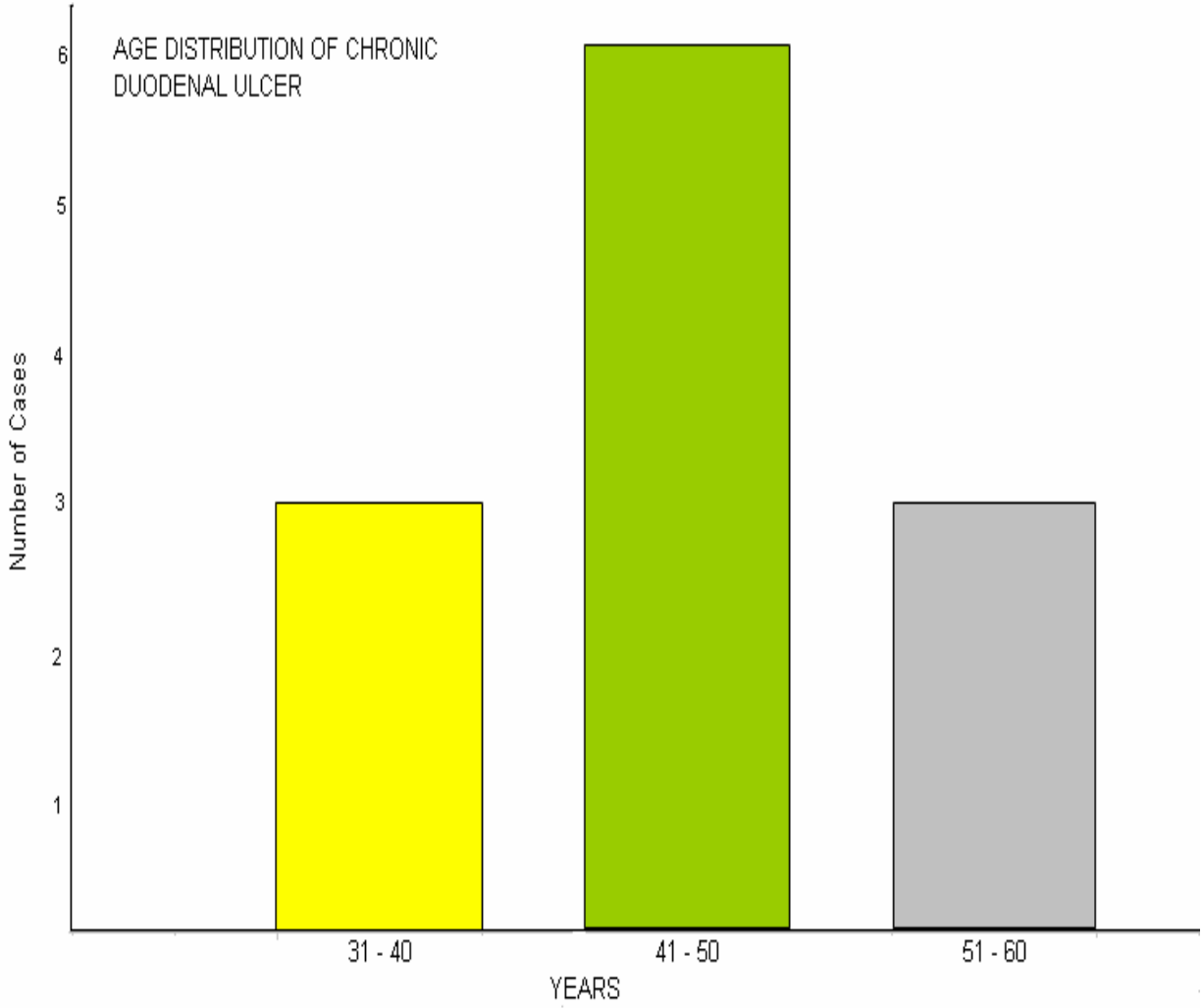


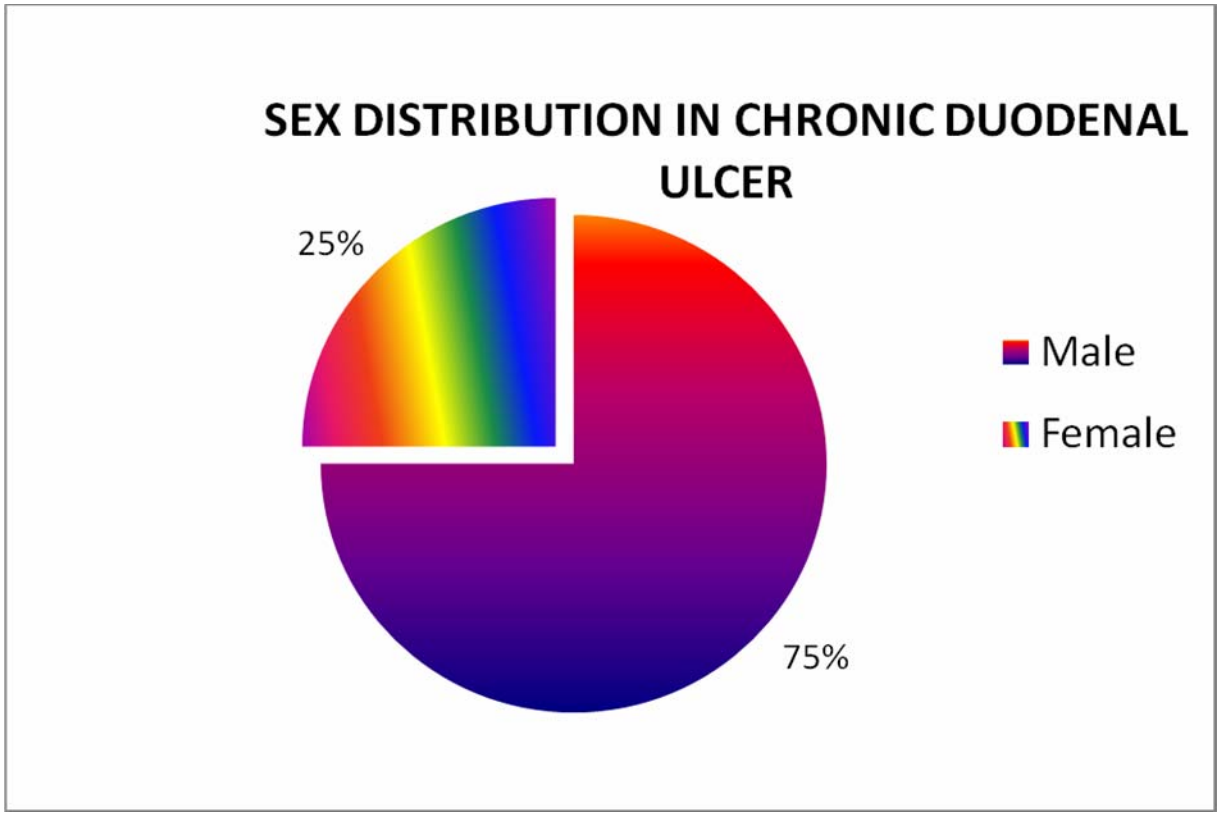
**RESECTED SPECIMEN OF CARCINOMA STOMACH**

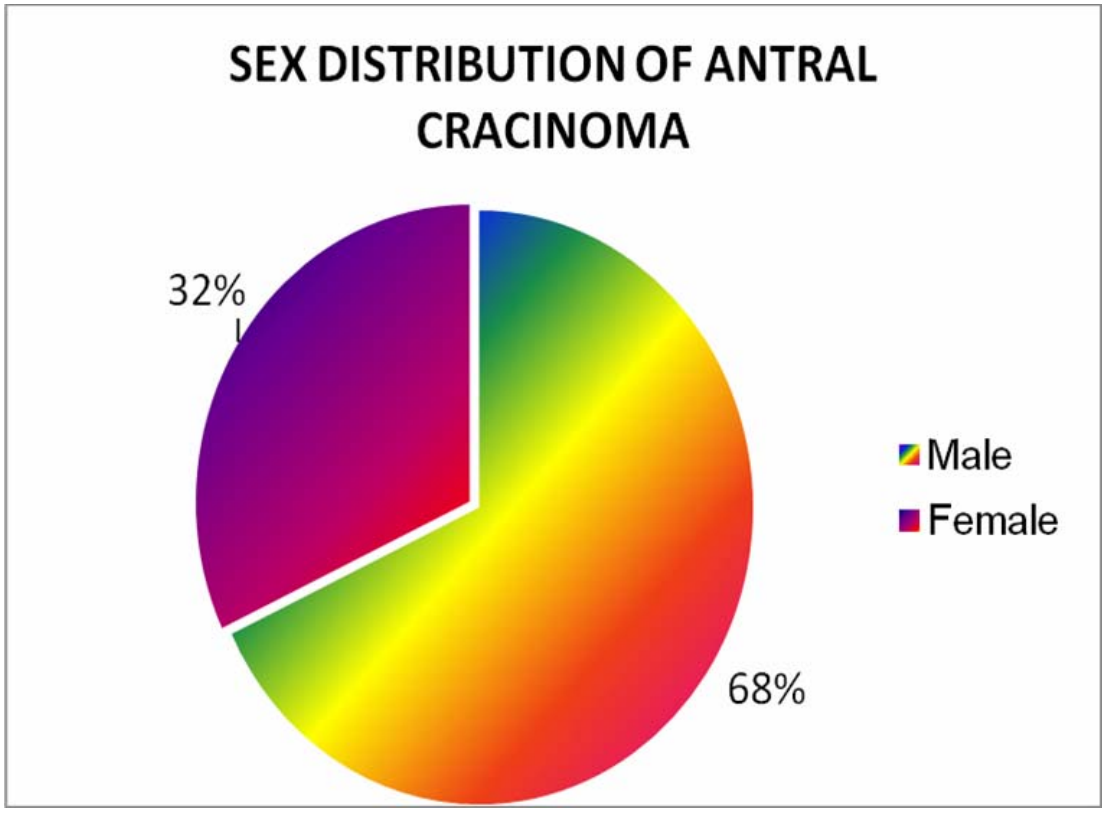



CAUSES OF GASTRIC OUTLET OBSTRUCTION IN ADULTS
Number of cases
50
45
40
35
30
25
20
15
10
5
Carcinoma of the antrum
Chronic duodenal Ulcer
Carcinoma head of Pancreas
CAUSES


AGE DISTRIBUTION OF ANTRAL CARCINOMA
Number of Cases
15
10
5
8 Cases
9 Cases
13 Cases
6 Cases
1 Case
30 - 40
41- 50
51- 60
61-70
71- 80
Years


AGE DISTRIBUTION OF CHRONIC DUODENAL ULCER
Number of Cases
6
5
4
3
2
1
31 - 40
41 - 50
51 - 60
YEARS


SEX DISTRIBUTION IN CHRONIC DUODENAL ULCER
25%
75%
Male
Female


SEX DISTRIBUTION OF ANTRAL CRACINOMA
32%
68%
Male
Female

# *BIBLIOGRAPHY*

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| S.No | Name | Age | Sex | IP.No | Epigastric pain | Vomitting | Anaemia | VGP | Mass | Liver metastasis | Ascites | OGD Diagnosis | Treatment | Post Op Period |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Manjula | 45 | F | 14450 | + | + | - | + | + | + | + | Antral growth | AGJ | Uneventful |
| 2 | Sampoornam | 45 | F | 11120 | + | + | - | + | + | + | - | Antral growth | AGJ | Uneventful |
| 3 | Vatchala | 50 | F | 11189 | + | + | - | + | + | + | - | Antral growth | AGJ | Uneventful |
| 4 | Lakshmi | 40 | F | 11109 | + | + | - | + | + | + | - | Antral growth | AGJ | Uneventful |
| 5 | Chinnaponnu | 60 | F | 1272 | + | + | + | + | + | + | - | Antral growth | AGJ | Uneventful |
| 6 | Natarajan | 55 | M | 918 | + | + | + | + | + | + | - | Antral growth | AGJ | Biliary Gastritis |
| 7 | Puroshothaman | 50 | M | 2230 | + | + | - | - | + | + | + | Antral growth | AGJ | Uneventful |
| 8 | Gowri | 70 | F | 21256 | + | + | - | + | + | + | - | Antral growth | AGJ | Uneventful |
| 9 | Subramani | 62 | M | 18160 | + | + | + | - | + | + | - | Antral growth | AGJ | Uneventful |
| 10 | Chandrasekar | 55 | M | 21059 | + | + | - | + | + | + | - | Antral growth | AGJ | Uneventful |
| 11 | Karuppaiah | 70 | M | 21280 | + | + | - | + | + | + | - | Antral growth | AGJ | Uneventful |
| 12 | Marimuthu | 60 | M | 18011 | + | + | - | - | + | + | + | Extraneous compression of | AGJ | Wound infection |
| 13 | Moorthy | 47 | M | 26204 | + | + | - | + | - | - | - | Antral growth | STG Billroth II | Uneventful |
| 14 | Masthan | 60 | M | 6541 | + | + | + | - | - | - | - | Antral growth | STG Billroth II | Uneventful |
| 15 | Seethapathy | 45 | M | 12658 | + | + | - | + | - | - | - | Antral growth | STG Billroth II | Uneventful |
| 16 | Lakshmi | 40 | F | 16147 | + | + | - | + | - | - | - | Antral growth | STG Billroth II | Uneventful |
| 17 | Ganesan | 37 | M | 22850 | + | + | - | + | - | - | - | Antral growth | STG Billroth II | Uneventful |
| 18 | Loorthuswamy | 57 | M | 2796 | - | + | - | + | + | - | - | Antral growth | STG Billroth II | Uneventful |
| 19 | Devaraj | 51 | M | 2773 | + | + | - | + | - | - | - | Antral growth | STG Billroth II | Uneventful |
| 20 | Jayanthi | 47 | F | 3456 | + | + | - | + | - | - | - | Antral growth | STG Billroth II | Uneventful |
| 21 | Mayan | 70 | M | 3267 | + | + | - | + | - | - | - | Antral growth | STG Billroth II | Uneventful |
| 22 | Baby | 40 | F | 21398 | + | + | - | + | - | - | - | Antral growth | STG Billroth II | Uneventful |
| 23 | Manikam | 65 | M | 26458 | + | + | - | + | + | - | - | Antral growth | STG Billroth II | Uneventful |
| 24 | Jayapaul | 38 | M | 25248 | + | + | - | + | - | - | - | Antral growth | STG Billroth II | Uneventful |
| 25 | Ganesan | 37 | M | 2455 | + | + | - | + | - | - | - | Antral growth | STG Billroth II | Bilious Vomiting |
| 26 | Padma | 40 | F | 5354 | + | + | + | + | + | - | - | Antral growth | STG Billroth II | Uneventful |

| | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 27 | Kumaravel | 43 | M | 7016 | + | + | - | - | + | - | - | Antral growth | STG Billroth II | Uneventful |
| 28 | Varadha kutty | 53 | M | 10196 | + | + | - | + | - | - | - | Antral growth | STG Billroth II | Uneventful |
| 29 | Alamelu | 60 | F | 11942 | + | + | - | - | + | - | - | Antral growth | STG Billroth II | Wound dehiscence |
| 30 | Poongavanam | 37 | M | 22401 | + | + | + | + | - | - | - | Antral growth | STG Billroth II | Uneventful |
| 31 | Mani | 60 | M | 10674 | + | + | - | + | + | - | - | Antral growth | STG Billroth II | Uneventful |
| 32 | Alwar | 70 | M | 5603 | + | + | + | + | - | - | - | Antral growth | STG Billroth II | Uneventful |
| 33 | Balakrishnan | 60 | M | 20818 | + | + | - | - | + | - | - | Antral growth | STG Billroth II | Uneventful |
| 34 | Kumar | 55 | M | 21366 | + | + | - | + | - | - | - | Antral growth | STG Billroth II | Uneventful |
| 35 | Mani | 45 | M | 20190 | + | + | - | + | - | - | - | Antral growth | STG Billroth II | Uneventful |
| 36 | Varathan | 60 | M | 16054 | + | + | - | + | - | - | - | Antral growth | STG Billroth II | Uneventful |
| 37 | Panchacharam | 80 | M | 18069 | + | + | + | + | - | - | - | Antral growth | STG Billroth II | Uneventful |
| 38 | Ravanamal | 55 | F | 18021 | + | + | - | - | - | - | - | Antral growth | STG Billroth II | Uneventful |
| 39 | krishnaveni | 40 | F | 35298 | + | + | - | + | - | - | - | Deformed DU bulb | TV + GJ | Uneventful |
| 40 | Amirthavalli | 50 | F | 7210 | + | + | - | - | - | - | - | Deformed DU bulb | TV + GJ | Uneventful |
| 41 | Narayanan | 50 | M | 11127 | + | + | - | + | - | - | - | Deformed DU bulb | TV + GJ | Uneventful |
| 42 | Gunasekaran | 45 | M | 16951 | + | + | - | + | - | - | - | Deformed DU bulb | TV + GJ | Wound dehiscence |
| 43 | kalakandan | 45 | M | 21260 | + | + | - | + | - | - | - | Deformed DU bulb | TV + GJ | Uneventful |
| 44 | Masii | 49 | M | 24659 | + | + | - | + | - | - | - | Deformed DU bulb | TV + GJ | Uneventful |
| 45 | Sambandan | 58 | M | 3207 | + | + | - | + | - | - | - | Deformed DU bulb | TV + GJ | Wound dehiscence |
| 46 | Balan | 35 | M | 32289 | + | + | - | + | - | - | - | Deformed DU bulb | TV + GJ | Uneventful |
| 47 | Vasantha | 55 | F | 16710 | + | + | - | - | - | - | - | Deformed DU bulb | TV + GJ | Uneventful |
| 48 | Kuppusamy | 55 | M | 27456 | + | + | - | + | - | - | - | Deformed DU bulb | TV + GJ | Uneventful |
| 49 | Veerakasi | 35 | M | 4235 | + | + | - | + | - | - | - | Deformed DU bulb | TV + GJ | Uneventful |
| 50 | Raju | 42 | M | 13367 | + | + | - | + | - | - | - | Deformed DU bulb | TV + GJ | Uneventful |